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SER. II. VOL. 19 NO. 3

TRANSACTIONS
OF
THE NEW YORK ACADEMY OF SCIENCES

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The Sections and the Division hold meetings regularly, one evening each month, during the academic year, October to May, inclusive. All meetings are held at the building of The New York Academy of Sciences, 2 East Sixty-third Street, New York 21, New York.

Conferences are also held at irregular intervals at times announced by special programs.

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TRANSACTIONS OF THE NEW YORK ACADEMY OF SCIENCES

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Executive Director: EUNICE THOMAS MINER

IN MEMORIAM

Benjamin Minge Duggar (1872-1956)

Dr. Benjamin Minge Duggar, Emeritus Professor of the University of Wisconsin, and Research Scientist at Lederle Laboratories, Pearl River, N. Y., died on September 10, 1956.

Dr. Duggar, the son of Reuben Henry Duggar and Margaret Louisa (Minge) Duggar, was born on September 1, 1872, in Gallion, Ala. At the age of 19, he received his Bachelor of Science degree from Mississippi Agricultural and Mechanical College and, in the following year, 1892, took his Master of Science degree at Alabama Polytechnic Institute. His other degrees include a Bachelor and Master of Arts from Harvard University, and a doctorate from Cornell, as well as honorary doctorates in science from the University of Wisconsin and from Washington University and of laws from the University of Missouri. He was an honorary Fellow of the International College of Surgeons.

In keeping with his philosophy that maintenance of mental and physical activity is the key to a long and satisfying life, Dr. Duggar vigorously pursued a full-time course of teaching, travel, and research. In 1895 he became assistant botanist at the State Laboratory of Natural History in Illinois, and in 1896 he moved to Cornell University, where he remained for five years, rising from instructor in plant physiology to assistant plant pathologist and then, in 1900, to assistant professor. In 1899 he went to Europe to study botany with Wilhelm Pfeffer at Leipzig and with Georg Klebs at Halle, Germany. He returned to Cornell in the fall of 1900. The following year he joined the Bureau of Plant Industry, United States Department of Agriculture, as plant physiologist. In that capacity he took charge of the study of cotton diseases throughout the South and Southwest, and went to Europe to study practical methods of mushroom culture. This latter step marked the beginning of experimental work that ultimately resulted in the introduction of pure-culture methods and of scientific methods in general in the mushroom growing industry in the United States.

After five years as professor of botany and head of the Department of Botany at the University of Missouri and five years as professor of plant physiology at the College of Agriculture, Cornell University, Dr. Duggar became research professor of plant physiology at Washington University, from 1912 to 1927. During this period he spent the year 1905-1906 studying abroad at the Botanical Institutes of the Universities of Munich and of Montpellier, and the years 1917-1919 as acting professor of biological chemistry at the Washington University Medical School. During the latter two years, Dr. Duggar substituted for P. A. Shaffer.

In 1927 Dr. Duggar went to the University of Wisconsin where he pursued his researches and taught until 1943. He was then one year older

than the age required for retirement by the state retirement law. He retired and was appointed emeritus professor.

In March 1944, at the invitation of the late Y. SubbaRow, Dr. Duggar joined the Lederle Laboratories Division of the American Cyanamid Company, Pearl River, N. Y., where he spent the last fourteen productive years of his active and busy life. It was here that, in September of 1945, he isolated, tested, and named the first strain of *Streptomyces aureofaciens*, the golden mold from which aureomycin was eventually obtained. Thus, still following his lifetime philosophy of keeping active, he found his greatest fame at the age of 75. Until his death he carried on a full-time research program, constantly searching for even more effective antibiotic drugs.

Well loved by his fellow workers, Dr. Duggar embodied all those qualities that he once said were the necessary human elements in successful research: "intellectual curiosity, adventurous spirit, critical interpretation, alertness to inferences, and steadfastness of faith and confidence." Much too modest to have referred to himself, he nevertheless summed up his own characteristics in these words.

Many distinctions came to Dr. Duggar during his lifetime; he was a member of the National Academy of Sciences, the American Philosophical Society, and the Philadelphia Academy of Science; trustee emeritus of the Marine Biological Laboratory at Woods Hole, Mass.; Fellow of the American Association for the Advancement of Science and Vice-President (Section G, Botany, 1927); honorary member of the Società Italiana di Ematologia; member of the Société Botanique de France; corresponding member of La Società Lancisiana, Rome, Italy; chairman of the Division of Mycology, The New York Academy of Sciences and, for three years, 1952 to 1956, member of the Council of The New York Academy of Sciences; member of the board of trustees, IBEC (International Basic Economy Corporation) Research Institute; member of the Society of Industrial Microbiology (President 1951 to 1952); member of the Botanical Society of America (President 1923); member of the American Society of Plant Physiology (President 1946 to 1947); member of the American Phytopathological Society, the American Chemical Society, the Society of American Bacteriologists, and the National Research Council (chairman, Division of Biology and Agriculture, 1925 to 1926, also chairman of the Committee on the Effects of Radiation on Organisms, 1935 to 1940); chairman of the Organizing Committee, also general secretary and chairman of the Executive Committee, International Congress of Plant Sciences, 1926; and a member of Sigma Xi, Phi Beta Kappa, and Phi Sigma.

Dr. Duggar was the author of more than one hundred articles in scientific journals and bulletin series. He wrote articles on fungi, fungous diseases of plants, physiology of the fungi, physiology of green plants, biological effects of radiation, and photosynthesis. He published three

books in his field, edited four others, and collaborated on a *Textbook of General Botany*.

The Council of The New York Academy of Sciences records with greatest regret the loss of so worthy a member. To his widow, Elsie Rist Duggar, to his children, and to his grandchildren, heartfelt sympathy is extended.

Prepared and read by
Hilary Koprowski
October 25, 1956

REPORT OF THE ANNUAL MEETING

December 6, 1956

The 139th Annual Meeting of the Academy, for the election of Fellows, the presentation of reports, and the transaction of other business, was held at the Academy Building on the evening of Thursday, December 6, 1956.

The first order of business was devoted to the reports of the Officers and Committees of the Academy.

REPORTS OF OFFICERS AND COMMITTEES

Report of the Recording Secretary. The benefit of preparing an annual report is that it gives the Officers of the Academy an opportunity to inform the Membership about the results achieved and to take an inventory of past performance, as well as to set new and higher levels to which the organization may be raised in the year ahead.

It should be apparent from the reports presented here that the Academy has just completed another year of successful endeavor with all aspects of its work giving assurance that in the year ahead, and in the further future, there are many promising possibilities for the Academy to help in expanding the horizon of science.

The achievements of the past year required considerable thought and effort on the part of a very large number of devoted persons, including the Council (your governing board composed of 38 Members), the Academy's regularly employed staff of 44, the 557 scientists who presented 444 papers at the regular meetings and the conferences or who participated in the organization of these meetings, and the more than 12,044 Members and many friends of the Academy who have supported its work by financial contribution or participation at its meetings.

The figures above give a total of more than 14,000 persons directly concerned with the successful performance of this society. This total does not include the untold thousands who make use of the publications and innumerable services that the Academy offers to all persons interested in scientific endeavor.

At this time we should express our deepest appreciation and thanks to these thousands of men and women who contribute their efforts, not only to the coordinated pattern of activities of this society, but also to the many-faceted constructive endeavors of life.

Seventeen conferences, listed below, were held during the year November 1955 through October 3, 1956.

"Some Protozoan Diseases of Man and Animals: Anaplasmosis, Babesiosis and Toxoplasmosis," November 17 and 18, 1955, Organizing Committee, Clarence R. Cole, Chairman, The Ohio State University, Columbus, Ohio; Hilary Koprowski, Lederle Laboratories Division, American Cyanamid Company, Pearl River, N. Y.; and Ross F. Nigrelli, New York Zoological Society, Bronx Park, New York, N.Y.

"Epidemic and Endemic Diarrheal Diseases of the Infant," December 8 and 9, 1955, Chairman, Erwin Neter, Children's Hospital, and University of Buffalo, Buffalo, N. Y.

"Calcium and Phosphorus Metabolism in Man and Animals with Special Reference to Pregnancy and Lactation," January 10 and 11, 1956, Organizing Committee, Robert R. Marshak, Chairman, Springfield, Vt.; and Robert L. Burkhart, Lederle Laboratories Division, American Cyanamid Company, Pearl River, N. Y.

"Second Tissue Homotransplantation Conference," February 2 and 3, 1956, Co-Chairmen, John Marquis Converse, New York University College of Medicine, New York, N. Y.; and Blair O. Rogers, St. Barnabas Hospital, Newark, N. J.

"Staphylococcal Infections," February 16 and 17, 1956, Chairman, David E. Rogers, Cornell University Medical College, New York, N. Y.

"The Electrophysiology of the Heart," February 16 and 17, 1956, Chairman, Hans H. Hecht, University of Utah, Salt Lake City, Utah.

"Natural Resistance to Infections," March 1 and 2, 1956, Organizing Committee, Rene Dubos, Chairman, Rockefeller Institute for Medical Research, New York, N. Y.; L. Pillemer, Institute for Pathology, Western Reserve Medical School, Cleveland, Ohio; and J. S. Kiser, Lederle Laboratories Division, American Cyanamid Company, Pearl River, N. Y.

"Experimental Methods for the Evaluation of Drugs in Various Disease States," March 8 and 9, 1956, Organizing Committee, Bradford N. Craver, Chairman, Squibb Institute for Medical Research, New Brunswick, N. J.; W. M. Govier, Secretary, Schering Corporation, Bloomfield, N. J.; Karl Beyer, Merck Sharp & Dohme, West Point, Pa.; K. K. Chen, Eli Lilly & Company, Indianapolis, Ind.; Raymond Cunningham, Lederle Laboratories Division, American Cyanamid Company, Pearl River, N. Y.; Edwin J. Fellows, Smith, Kline & French Laboratories, Philadelphia, Pa.; H. J. Robinson, Merck Institute for Therapeutic Research, Rahway, N. J.; Lowell Randall, Hoffmann-La Roche, Inc., Nutley, N. J.; Clement A. Stone, Merck Sharp & Dohme, West Point, Pa.; E. B. Robbins, Eli Lilly & Company, Indianapolis, Ind.; Edwin J. de Beer, Wellcome Research Laboratories, Tuckahoe, N. Y.; Hans Molitor, Merck Institute for Therapeutic Research, Rahway, N. J.; Albert J. Plummer, CIBA Pharmaceutical Products, Inc., Summit, N. J.; Richard K. Richards, Abbott Laboratories, North Chicago, Ill.; and Joseph Seifter, Wyeth Institute, Philadelphia, Pa.

"Unstable Chemical Species: Free Radicals, Ions, and Excited Molecules," March 15 and 16, 1956, Co-Chairmen, Thomas Freund, New York University, New York, N. Y.; and Henry C. Thacher, Jr., Aeronautical Research Laboratory, Wright Air Development Center, Ohio.

"Mercury and Its Compounds," April 6 and 7, 1956, Co-Chairmen, Cecil V. King, New York University, New York, N. Y.; Harry Gold, Cornell University Medical College, New York, N. Y.; and Harold Wolff, Harold Wolff Associates, New York, N. Y.

"The Pharmacology of Psychotomimetic and Psychotherapeutic Drugs," April 12, 13 and 14, 1956, Chairman, Seymour S. Kety, National Institute of Neurological Diseases and Blindness, National Institutes of Health, Bethesda, Md.

"Basic Problems in Geophysics," April 20, 1956, Committee of The New York Academy of Sciences for the International Geophysical Year 1957-1958: Co-Chairmen, Boris Pregel, The Canadian Radium and Uranium Corporation, New York, N. Y.; and Serge Korff, New York University, New York, N. Y.; Members, William O. Field, American Geographical Society, New York, N. Y.; Bernhard Haurwitz, New York University, New York, N. Y.; Charles Hitchcock, American Geographical Society; and Walter A. Wood, Arctic Institute of North America, New York, N. Y.

"Techniques for the Study of Behavioral Effects of Drugs," May 4, 1956, Co-Chairmen, Peter B. Dews, Harvard Medical School, Boston, Mass.; and B. F. Skinner, Harvard Medical School, Cambridge, Mass.

"Anesthesiology," May 18 and 19, 1956, Co-Chairmen, C. R. Stephen, Duke University School of Medicine, Durham, N. C.; and E. J. de Beer, Wellcome Research Laboratories, Tuckahoe, N. Y.

"Viruses in Search of Disease," May 24 and 25, 1956, Chairman, Robert J. Huebner, Laboratory of Infectious Diseases, National Institute of Allergy and Infectious Diseases, National Institutes of Health, Bethesda, Md.

"Man-Made Fiber Progress," October 12, 1956, Chairman, J. J. Press, Polytechnic Institute of Brooklyn, and United States Navy Clothing Supply Office, Brooklyn, N. Y.

"Meprobamate and Other Agents Used in Mental Disturbances," October 18 and 19, 1956, Co-Chairmen, James G. Miller, Mental Health Research Institute, University of Michigan, Ann Arbor, Mich.; and Frank M. Berger, Medical Director, Wallace Laboratories, New Brunswick, N. J.

This is not only the largest series of conferences yet held by the Academy in a single year but, in keeping with the Academy's aim to encourage closer communication of scientists of all disciplines with one another, the programs, as developed, include a broad cross section of many fields of research.

The Council is profoundly grateful for the active interest and resolute manner with which these conferences are developed by investigators, without regard for

competition in achievement, but rather with the determination freely to present, discuss, and publish the results of research for their broadest possible dissemination.

Exceptional thanks are extended to those persons, both Members and non-members of the Academy, who have given so generously of their time, energy, and knowledge in developing these conferences and thus furthering this phase of the Academy's activities.

Three hundred and eighty-seven meetings were held in the Academy Building during the past year. These covered not only Sectional meetings, conferences, Committee and Council meetings of the Academy, but 276 were those of 51 other societies to which the accommodations of the building were extended.

This series of reports for the fiscal year 1955-1956 presents a summary of some of the major achievements of the Academy during this year and should bring satisfaction to the Members of the Academy and to all others interested in the advancement of science as an integral contribution toward the betterment of human welfare.

The Academy is continuing to expand its Membership. During this year ending October 31, 1956, 2,791 new Members were added to our rolls, as follows: 6 Honorary Life Members; 9 Active Life Members; 96 Sustaining Members; 2,659 Annual Active Members; and 21 Student Members. Seventy-one former Members were reinstated, thus making a gross total of 2,862 names added to the current membership rolls. Twenty-three transfers were effected in the Annual Classes and 3 Annual Active Members were transferred to Active Life Membership.

The Academy lost by death 2 Honorary Life Members; 4 Active Life Members; 4 Sustaining Members; and 52 Annual Active Members. Three hundred and twenty-two resignations were accepted and 365 Members were dropped as not having paid dues or for not having qualified for membership.

The Academy now stands with a net gain of 2,113 Members for the current year. There are at present upon the rolls of the Academy 12,044 Members, of whom 1,132 are Fellows, classified as follows:

3 Benefactors	479 Sustaining Members
2 Patrons	11,008 Active Members
62 Honorary Life Members	89 Student Members
394 Active Life Members	7 Corporation Members

Record is made with regret of the loss by death of the following Members:

Active Life Members

Henry E. Crampton, elected to Active Membership, 1894; transferred to Life Membership, 3 February, 1919. Died 26 February, 1956. Fellow. Recording Secretary and Vice-President, 1906; Corresponding Secretary, 1908-1925; President, 1926.

Adolph Elwyn, elected to Associate Membership, 6 March, 1916; transferred to Active Membership, 3 December, 1917; transferred to Life Membership, 23 April, 1936. Died June, 1955. Fellow.

Roy Waldo Miner, elected to Active Membership, January, 1905; transferred to Life Membership, 6 October, 1909. Died 13 December, 1955. Fellow. Assistant Recording Secretary, 1905; Recording Secretary, 1926-1937; Corresponding Secretary, 1939; President, 1940, 1941; Editor, 1943-1955.

Nathaniel W. Winkelman, elected to Sustaining Membership, 18 November, 1948; transferred to Life Membership, 10 March, 1950. Died 13 February, 1956.

Sustaining Members

Ernst A. Hauser, elected to Sustaining Membership, 12 June, 1941. Died 10 February, 1946. Fellow.

Harold Houston Jones, elected to Sustaining Membership, 28 October, 1954. Died, 1956.

O. P. Robinson, Jr., elected to Sustaining Membership, 15 June, 1953. Died 27 February, 1956.

Simonide G. Vlavianos, elected to Sustaining Membership, 25 October, 1951. Died August, 1956.

Active Members

Mark H. Adams, elected to Active Membership, 4 November, 1940. Died 18 October, 1956. Fellow.

William H. Bauer, elected to Active Membership, 26 January, 1956. Died 14 June, 1956.
Louis Bauman, elected to Active Membership, 14 May, 1948. Died 1 November, 1954.
Samuel S. Berger, elected to Active Membership, 23 September, 1954. Died 18 September, 1956.
Angelo E. Benaglia, elected to Active Membership, 26 February, 1942. Died 3 March, 1956.
George C. Bishop, elected to Active Membership, 23 September, 1954. Died 15 January, 1956.
Albert E. Bothe, elected to Active Membership, 15 August, 1949. Died 11 November, 1955.
Aaron S. Cantor, elected to Active Membership, 18 August, 1954. Died 18 April, 1955.
Mortimer Danzer, elected to Active Membership, 20 November, 1952. Died 5 September, 1956.
Benjamin Minge Duggar, elected to Active Membership, 15 February, 1945. Died 10 September 1956. Fellow. Councilor 1953-1956.
Maxwell J. Fein, elected to Active Membership, 20 November, 1952. Died 14 July, 1955.
Joseph Felsen, elected to Active Membership, 15 July, 1952. Died July, 1956.
Harold C. Fischer, elected to Active Membership, 21 June, 1956. Died 27 June, 1956.
Alexander Frieden, elected to Active Membership, 6 May, 1940. Died September, 1956.
Jonas S. Friedenwald, elected to Active Membership, 22 April, 1954. Died 5 November, 1955.
John C. Garey, elected to Active Membership, 15 May, 1952. Died 16 August, 1956.
S. B. Grimson, elected to Active Membership, 13 July, 1951. Died 29 November, 1955.
H. Randolph Halsey, elected to Section Membership, 4 February, 1929. Resigned, 26 January, 1939. Reinstated, Active Membership, 3 February, 1941. Died 23 December, 1955.
George Heller, elected to Active Membership, 15 May, 1947. Died 28 December, 1955.
Fred W. Holmes, elected to Active Membership, 27 October, 1949. Died 8 August, 1955.
Alfred E. Johns, elected to Active Membership, 19 November, 1953. Died 31, July, 1956.
Henry L. Kirkendall, elected to Active Membership, 28 April, 1955. Died 8 May, 1956.
Abner Kurtin, elected to Active Membership, 15 July, 1952. Died August, 1956.
Joseph L. Lilienthal, Jr., elected to Active Membership, 27 February, 1947. Died November, 1955. Fellow.
Paul Loewy, elected to Active Membership, 28 April, 1949. Died 9 December, 1955.
Ellice McDonald, elected to Active Membership, 4 December, 1939. Died 30 January, 1955.
Howard M. Marjerison, elected to Active Membership, 17 May, 1954. Died 4 September, 1955.
Boris Mirkine-Guetzevitch, elected to Active Membership, 30 June, 1954. Died 1 April, 1955.
Louis Nerb, elected to Active Membership, 3 February, 1941. Died 19 May, 1955.
Helen M. Noyes, elected to Active Membership, 2 December, 1940. Died October, 1955.
Harry W. Orr, elected to Active Membership, 26 April, 1951. Died 14 January, 1956.
Clayton Roberts Orton, elected to Active Membership, 15 May, 1952. Died 17 June, 1955.
Beryl H. Paige, elected to Active Membership, 3 April, 1939. Died March, 1956.
Alwin M. Pappenheimer, elected to Active Membership, 2 October, 1916. Died 21 February, 1955. Fellow.
Robert K. Phelan, elected to Active Membership, 13 August, 1953. Died 17 November, 1955.
Sylvester Pincus, elected to Active Membership, 20 December, 1955. Died, 1956.
Bo Prytz, elected to Active Membership, 15 May, 1947. Died May, 1956.
Pierre J. Rabil, elected to Active Membership, 26 April, 1956. Died September, 1956.
Robert R. Redfield, elected to Active Membership, 17 November, 1955. Died January, 1956.
L. Reiner, elected to Active Membership, 4 March, 1940. Died 27 November, 1955.
James F. Rinehart, elected to Active Membership, 28 April, 1955. Died 30 November, 1955.
Maurycy Saidman, elected to Active Membership, 26 March, 1953. Died August, 1956.
Arthur F. St. Andre, elected to Active Membership, 23 January, 1947. Died, 1955.
Salvatore M. Santelli, elected to Active Membership, 23 October, 1952. Died, 1955.

John A. Saxton, Jr., elected to Active Membership, 27 October, 1949. Died February, 1956.
Ephraim Shorr, elected to Active Membership, 14 June, 1946. Died 6 January, 1956. Fellow.
Gershon B. Silver, elected to Active Membership, 22 January, 1948. Died 22 August, 1955.
Kurt G. Stern, elected to Active Membership, 4 March, 1940. Died 3 February, 1956. Fellow.
Motohiko Tobicka, elected to Active Membership, 27 January, 1955. Died 26 January, 1956.
Howard Walton, Jr., elected to Active Membership, 15 July, 1955. Died November, 1955.
Jacob Werne, elected to Active Membership, 14 June, 1946. Died 14 April, 1955. Fellow.
Adolf Zeckel, elected to Active Membership, 14 September, 1951. Died 17 March, 1955

Report of the Corresponding Secretary. There are, at present, upon the rolls of the Academy, 62 Honorary Life Members. The Corresponding Secretary takes this opportunity of presenting the Members of the Academy with an up-to-date list of the Honorary Life Membership Class:

Sven Olof Asplund ..... Gothenburg, Sweden
William Thomas Astbury ..... Leeds, England
Ernst David Bergmann ..... Tel-Aviv, Israel
Charles Herbert Best ..... Toronto, Canada
Niels Bjerrum ..... Copenhagen Valby, Denmark
Niels Bohr ..... Copenhagen, Denmark
Henri Breuil ..... Paris, France
MacFarlane Burnet ..... Victoria, Australia
Adolf F. J. Butenandt ..... Tubingen, Germany
M. Albert Caquot ..... Paris, France
Alfonso Caso ..... Mexico, D.F., Mexico
Maurice Caullery ..... Paris, France
J. A. Christiansen ..... Copenhagen, Denmark
James Bertram Collip ..... London, Canada
Arthur H. Compton ..... St. Louis, Missouri
Eduardo Cruz-Coke ..... Santiago, Chile
Henrique Da Rocha Lima ..... Sao Paulo, Brazil
Sir Henry Dale ..... London, England
Thomas Dalling ..... Rome, Italy
Clinton J. Davisson ..... Charlottesville, Virginia
Peter J. W. Debye ..... Ithaca, New York
Albert Defant ..... Innsbruck, Austria
Ulick R. Evans ..... Cambridge, England
Emmanuel Faure-Fremiet ..... Paris, France
Howard Walter Florey ..... Oxford, England
Ross Granville Harrison ..... New Haven, Connecticut
David Kennedy Henderson ..... Edinburgh, Scotland
Corneille J. F. Heymans ..... Ghent, Belgium
Bernardo A. Houssay ..... Buenos Aires, Argentina
Julian S. Huxley ..... London, England
Christopher Kelk Ingold ..... London, England
Harold Jeffreys ..... Cambridge, England
Peter Kapitza ..... Moscow, U.S.S.R.
Vasant Ramj Khanolkar ..... Bombay, India
Alfred L. Kroeber ..... Berkeley, California
Hugo Rudolph Kruyt ..... The Hague, Holland
Irving Langmuir ..... Schenectady, New York
K. S. Lashley ..... Orange Park, Florida
Kaj U. Linderstrom-Lang ..... Copenhagen Valby, Denmark
Robert H. Lowie ..... Berkeley, California
Andre Lwoff ..... Paris, France
Vladimir A. Obruchev ..... Moscow, U.S.S.R.
Marcus L. E. Oliphant ..... Canberra, Australia
Jean Piaget ..... Geneva, Switzerland
Henri Pieron ..... Paris, France
A. R. Radcliffe Brown ..... Grahamstown, South Africa
Gaston Leon Ramon ..... Paris, France
Sir Robert Robinson ..... Great Missenden, Bucks, Eng.
C. G. Rossby ..... Stockholm, Sweden
John A. M. Runnstrom ..... Stockholm, Sweden
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Arne Tiselius ..... Uppsala, Sweden
Felix A. Vening-Meinesz ..... Anerafoort, Holland
Henri Rene Villat ..... Paris, France
Max Von Laue ..... Berlin, Germany
D. M. S. Watson ..... London, England
Rudolf Weigl ..... Cracow, Poland

Record is made with regret of the death of the following Honorary Life Members:

Oswald T. Avery, Nashville, Tennessee, elected December 15, 1943, died February 20, 1955.
Albert Jan Kluyver, Delft, Holland, elected December 7, 1949, died May 14, 1956.

Report on the Status of Publications. During the fiscal year ending October 31, 1956, The New York Academy of Sciences has published 24 monographs, comprising a total of 2591 pages. Included in these are 226 papers by 331 authors. The pagination of the *Transactions* (768) brings the total pagination issued to 3359 pages. The list of publications issued during the fiscal year is as follows:

ANNALS

Volume 62, Article 14. *Effect of Malonate on Growth Rate of* Salmonella typhimurium *in Mice* (1 paper), by L. Joe Berry. Pages 327-348. Published November 2, 1955.
Volume 62, Article 15. *Nutritional Factors in Thermophily: A Comparative Study of Bacilli and* Euglena (1 paper), by Herman Baker *et al.* Pages 349-376. Published November 21, 1955.
Volume 62, Article 16. *A Hypothesis for a Mechanism of Cardiac Glycoside Action* (1 paper), by Charles D. Proctor *et al.* Pages 377-402. Published December 29, 1955.
Volume 62, Article 17. *Quantitative Relations Between Liver Mitochondria Metabolism and Total Body Weight in Mammals* (1 paper), by Robert E. Smith. Pages 403-422. Published January 31, 1956.
Volume 62, Article 18. *The Spin of Thorium C′* (1 paper), by Arthur Beiser. Pages 423-434. Published February 3, 1956.
Volume 62, Article 19. *Lead Isotopes as a Key to the Radioactivity of the Earth's Mantle* (1 paper), by R. D. Russell. Pages 435-448. Published February 6, 1956.
Volume 62, Article 20. *An X-Ray Study of Uraninite* (1 paper), by William J. Croft. Pages 449-502. Published February 10, 1956.
Volume 62, Article 21. *Extinct Natural Radioactivity: Possibilities and Potentialities* (1 paper), by Truman P. Kohman. Pages 503-542. Published April 13, 1956.
Volume 63, Article 3. *Psychotherapy and Counseling* (18 papers), by L. Frank *et al.* Pages 321-432. Published November 7, 1955.
Volume 63, Article 4. *Dynamic Anthropometry* (16 papers), by Earl W. Count *et al.* Pages 433-636. Published November 28, 1955.
Volume 63, Article 5. *Ascites Tumors as Tools in Quantitative Oncology* (30 papers), by Theodore S. Hauschka *et al.* Pages 637-1030. Published March 14, 1956.
Volume 63, Article 6. *Cancer Cytology and Cytochemistry* (36 papers), by M. J. Kopac *et al.* Pages 1033-1462. Published March 30, 1956.
Volume 64, Article 1. *The Organization of The New York Academy of Sciences. Its Incorporation, Its Amended Charter, Its Constitution and Bylaws.* Revised to November 17, 1955. Pages 1-24. Published April 26, 1956.
Volume 64, Article 2. *Some Protozoan Diseases of Man and Animals: Anaplasmosis, Babesiosis, and Toxoplasmosis* (18 papers), by C. C. Cole *et al.* Pages 25-277. Published July 5, 1956.
Volume 64, Article 3. *Calcium and Phosphorus Metabolism in Man and Animals with Special Reference to Pregnancy and Lactation* (18 papers), by F. C. McLean *et al.* Pages 279-462. Published August 17, 1956.
Volume 65, Article 1. *Effects of Natural Selection on Human Genotypes* (1 paper), by L. D. Dunn *et al.* Pages 1-32. Published June 18, 1956.
Volume 65, Article 2. *On the Cell Model for Solutions* (1 paper), by Stuart A. Rice. Pages 33-54. Accorded an A. Cressy Morrison Award in Natural Science in 1955. Published June 21, 1956.
Volume 65, Article 3. *Staphylococcal Infections* (21 papers), by David E. Rogers *et al.* Pages 57-246. Published August 31, 1956.
Volume 66, Article 1. *Epidemic and Endemic Diarrheal Diseases of the Infant* (33 papers), by James A. Baker *et al.* Pages 3-230. Published August 10, 1956.
Volume 66, Article 2. *Natural Resistance to Infections* (21 papers), by Walsh McDermott *et al.* Pages 233-441. Published October 5, 1956.
Volume 67, Article 1. *Diet Selections of Rats Subjected to Stress* (1 paper), by William J. Griffiths, Jr. Pages 1-10. Published June 26, 1956.
Volume 67, Article 2. *The Effects of Continuous Irradiation by Tritium on Cells Cultivated in Vitro* (1 paper), by Agnes N. Stroud. Pages 11-34. Accorded an A. Cressy Morrison Award in Natural Science in 1955. Published August 29, 1956.
Volume 67, Article 3. *The Effects of Cosmic Rays on the Terrestrial Isotope Distribution* (1 paper), by Serge A. Korff. Pages 35-54. Accorded The Boris Pregel Award for Research on Natural Radioactive Substances in 1955. Published September 4, 1956.
Volume 67, Article 4. *Cytology and Cytochemistry of Melanoma Cells* (1 paper), by Sylvia S. Greenberg *et al.* Pages 55-122. Published October 11, 1956.

In addition to the publications cited above, the following monographs were published during November 1956, after the close of the fiscal year.

Volume 64, Article 4. *Experimental Methods for the Evaluation of Drugs in Various Disease States* (26 papers), by Bradford N. Craver *et al.* Pages 463-732. Published November 16, 1956.

Volume 65, Article 4. *Techniques for the Study of Behavioral Effects of Drugs* (8 papers), by Peter B. Dews *et al.* Pages 247-356. Published November 2, 1956.
Volume 67, Article 5. *Recent Application of Autonomic Responses: Toward a General Solution* (1 paper), by J. I. Lacey. Pages 123-164. Published November 30, 1956.

TRANSACTIONS

Series II, Volume 18, Numbers 1 to 8, consisting of 768 pages, covering the period from November 1955 to June 1956, inclusive, was completed, printed, and distributed during the calendar year.

ANNALS IN PRESS

Volume 64, Article 5. *Second Tissue Homotransplantation Conference* (30 papers), by John Marquis Converse *et al.* Now in galley proof.
Volume 65, Article 5. *Mercury and Its Compounds.* First galleys expected December 5, 1956.
Volume 66, Article 3. *The Pharmacology of Psychotomimetic and Psychotherapeutic Drugs.* Correction of galleys virtually completed.
Volume 66, Article 4. *Anesthesiology.* Monograph sent to the printer November 1, 1956. Galleys awaited.

From November 1, 1955 to October 31, 1956, the Academy distributed 304,266 separate publications and 20,620 complete volumes of the various series as follows:

	Separate Numbers	*Volumes*
Annals		
Members	90,764	
Gratis: authors, reviews, exchanges, and donors	12,400	
Sales	68,562	118
Authors' reprints	93,850	
Transactions		
Members	4,393	10,172
Gratis: authors, exchanges, and reviews	3,737	
Sales	248	268
Authors' reprints	16,835	
Special Publications, Volumes I, III, IV		
Sales		59
Scientific Survey of Porto Rico and the Virgin Islands		
Gratis	38	8
Sales	275	33
Monthly Program		
Members	8,347	9,962
Sales	3,223	
Gratis: individuals	1,594	
	304,266	20,620

The total *list* value of publications distributed to the members, and gratis to authors, donors, reviewers, and exchange institutions is $319,519.

Report of the Treasurer. This report of the Treasurer on the results of the financial operations of the Academy supplements the attached statement of the auditors covering the Balance Sheet of Assets and Liabilities, in addition to the Statement of Income and Expenses for the fiscal year November 1, 1955 to October 31, 1956. These statements reflect the closing figures at October 31, 1956, and compare them with those of the preceding year.

The capital and operating assets, combined, reached a new peak in 1956, with a total of $1,147,504.24 as at October 31, 1956. The net gain for the year is $71,569.28, after the deduction of $38,588.81 from the Fixed Assets of the Building Fund. This latter amount, formerly carried on the Balance Sheet as representing furniture, fixtures, and equipment has been deducted in conformity with the practice usually followed by similar organizations when provision is not made for depreciation of such Fixed Assets.

A total increase in assets of $110,158.09 for 1956 is credited into several categories of the Academy's accounts, as follows:

General operating funds	$ 4,334.49
Endowment funds	51,035.33
Building fund	3,300.00
Prize funds (income only)	680.09
Special funds	50,808.18
	$110,158.09

The Finance Committee, through its continued sustained efforts in the successful management of the Academy's investment portfolio, increased the Reserve Account for Capital Gains and Losses by the sum of $47,325.33.

In January, the Council changed the name of the Publication Fund to The Roy Waldo Miner Memorial Publication Fund as a fitting and permanent memorial to Doctor Miner in recognition of his many years of devoted service to the work of the Academy. To date the contributions from the Membership and the many friends of Doctor Miner have increased this fund by $3,710.00 so that it now totals $6,893.00. The income earned is to be applied toward defraying prime publication costs.

These additions to the Endowment Funds in 1956 have increased the total to $533,795.47, a net gain of nearly 10.5 per cent.

The increase of $3,300 in the Building Fund, resulting from 11 new Life Members, has been credited to its Investment and Pledges Receivable accounts. The income from these investments is applied to the general costs of building maintenance.

In the seven years that the Academy has held its building, it has reduced its mortgage by the sum of $47,017.91, with a balance due at October 31, 1956, of $152,902.09. In addition to regularly amortizing its mortgage through quarterly payments, the Academy has maintained the building in a splendid condition. During the past year, the kitchen and pantry have been completely renovated and modernized with excellent mechanized, stainless steel equipment at a cost of nearly $25,000.

The earned income of the general operating funds totaled $389,104.60, while the actual expenses amounted to $378,337.39, thus showing an excess of income over expenses of $10,767.21. This figure added to $6,988.26, the General Fund surplus as of October 31, 1955, provided a gross surplus of $17,755.47. After deduction of unexpended investment income of the Endowment Funds and of the equity of mortgage principal paid in 1956, the new General Fund surplus as of October 31, 1956, now stands at $5,791.11.

Contributions in aid of conferences totaled $63,008.55. Of this amount, $34,000.21 was spent to reimburse speakers for their traveling expenses; $9,915.46 aided the Academy in defraying the routine expenses of conferences; and $19,092.88 was applied toward the publication costs of conference monographs.

Deep appreciation and thanks are expressed to the donors of these grants-in-aid that help make possible the holding of the Academy's well-known series of conferences and the publication of the resulting monographs. This phase of the Academy's activities is worthy of receiving greater support. The major portion of the expenses of these conferences ($112,302.81 in 1956) is now borne by the income from Members' dues, and more aid from outside sources would mean increased benefits to the membership and to science.

This report on the financial status of the Academy reflects the creditable record of this society's activities during the past year. The gratifying results that have been achieved are due to the exceptional cooperation and enthusiastic endeavor of the Members, Officers, and the Staff, and the opportunities developing for the year 1957 offer a higher goal toward which we must aim for greater results.

The books of accounting have been checked and audited by the firm of Peat, Marwick, Mitchell & Co., Certified Public Accountants. The property of the Academy has been verified by the auditors and by the Finance Committee of the Academy.

The report of the auditors has been approved by the Finance Committee as of November 28, 1956, as provided in the Bylaws.

Report of the Auditors

PEAT, MARWICK, MITCHELL & CO.
CERTIFIED PUBLIC ACCOUNTANTS
SEVENTY PINE STREET
NEW YORK 5, N. Y.

Accountants' Report

To the Finance Committee
The New York Academy of Sciences
New York, N. Y.

We have examined the balance sheet of The New York Academy of Sciences as of October 31, 1956, and the related statement of General Fund income and expenses and surplus for the year then ended. Our examination was made in accordance with generally accepted auditing standards, and accordingly included such tests of the accounting records and such other auditing procedures as we considered necessary in the circumstances.

The cash has been confirmed by certificate obtained directly from the depositary or by count. The cash held by J. R. Williston & Co., carried for the purchase of securities, has been confirmed by acknowledgment from the custodian.

We did not confirm the members' dues or the pledges receivable by communication with the respective members or pledgers. We communicated with all debtors on miscellaneous accounts receivable, representing charges for publications and use of building, etc., by mailing the Academy's statements for the month of October accompanied by a request that we be notified of any exceptions. The exceptions reported were explained to our satisfaction. Provision has not been made for possible loss in collection of receivables.

The inventory of the printing division is stated in accordance with a physical inventory taken and valued by employees of the Academy and certified by the Executive Director as to description, quantities, condition, and valuation. We made tests of the quantities, prices, and computations. The balance sheet does not reflect the books and publications on hand at October 31, 1956, as it is the policy of the Academy to charge publication costs to expense as incurred.

The marketable securities were inspected and the income therefrom was confirmed by reference to independent sources.

We inspected the deed to the real estate at 2 East 63rd Street, New York, N.Y., contributed to the Academy in 1949. It is carried in the balance sheet at the valuation, $425,000.00, as assessed by the City of New York for real estate tax purposes as of the date of the contribution, plus the cost of improvements. The amount of $38,588.81 at which the furniture, fixtures, and equipment were heretofore carried in the Building Fund, representing the assigned value of $10,000.00 to furnishings contributed and purchased to October 31, 1953, was removed from the assets by deduction from the principal of the fund in 1956. The cost of equipment purchased since October 31, 1953, has been charged to the income of the General Fund. In conformity with the practice usually followed by similar organizations, the Academy does not provide for depreciation of the building and improvements.

All liabilities of which we obtained knowledge as a result of our examination have received appropriate recognition.

In our opinion, subject to the foregoing comments, the accompanying balance sheet and statement of General Fund income and expenses and surplus present fairly the financial position of The New York Academy of Sciences at October 31, 1956, and the results of its operations for the year then ended on a basis consistent with that of the preceding year.

/s/ Peat, Marwick, Mitchell & Co.

New York, N. Y.
November 21, 1956

THE NEW YORK ACADEMY OF SCIENCES

Balance Sheet

October 31, 1956 and 1955

ASSETS

	1956	*1955*
General Fund		
Cash	$ 13,159.77	$ 10,356.10
Receivables: Members' dues	7,605.00	6,455.00
Miscellaneous accounts	12,601.26	12,205.69
Inventory, printing division	5,150.23	5,303.38
Prepaid expenses	1,133.89	995.49
	$ 39,650.15	$ 35,315.66
Endowment Funds		
Cash deposited with J. R. Williston & Co.	$ 57,426.87	$ 21,898.68
Marketable securities – stated at purchase cost or quoted price at date of contribution (quoted market value at October 31, 1956, $495,868.43)	476,368.60	$460,861.46
	$533,795.47	$482,760.14
Building Fund		
Cash	$ 79.94	$ (410.51)
Receivables – pledges	1,422.50	437.50
Marketable securities – stated at purchase cost or quoted price at date of contribution (quoted market value at October 31, 1956, $16,037.50)	14,618.04	12,793.49
Real estate at 2 East 63rd Street, New York, N.Y., at assessed valuation at date of contribution, and improvements ($27,280.63) at cost	452,280.63	452,280.63
Furniture, fixtures, and equipment, contributed or purchased prior to October 31, 1953 (deducted from principal of fund in 1956)	–	38,588.81
	$468,401.11	$503,689.92
Prize Funds		
Cash	$ 2,888.38	$ 2,208.29
Marketable securities – stated at purchase cost or quoted price at date of contribution (quoted market value at October 31, 1956, $39,150.44)	16,546.79	16,546.79
	$ 19,435.17	$ 18,755.08
Special Funds		
Cash	$ 81,579.30	$ 35,324.16
Receivables – miscellaneous accounts	2,821.00	90.00
Due from endowment funds	1,822.04	–
	$ 86,222.34	$ 35,414.16

Balance Sheet — *Continued*

As of October 31, 1956 and 1955

LIABILITIES AND FUNDS

	1956	*1955*
General Fund		
Liabilities:		
Accounts payable	$ —	$ 33.80
Mortgage interest accrued, at rate of 4½% per annum	1,147.36	1,206.56
Members' dues unearned	30,056.68	24,762.04
Members' dues collected in advance	2,655.00	2,325.00
Fund surplus, per accompanying statement	5,791.11	6,988.26
	$ 39,650.15	$ 35,315.66
Endowment Funds		
Principal of funds:		
John James Audubon Fund	$ 2,500.00	$ 2,500.00
Nathaniel Lord Britton Fund	30,039.77	30,039.77
Centennial Endowment Fund	7,934.00	7,934.00
Roy Waldo Miner Memorial Publication Fund	6,893.00	3,183.00
John Strong Newberry Fund	1,000.00	1,000.00
George H. Sherwood Memorial Fund	7,000.00	7,000.00
Ralph Winfield Tower Memorial Fund	12,728.00	12,728.00
Thomas Lincoln Casey Fund	213,461.99	213,461.99
John Roger Fund	1,000.00	1,000.00
Capital gains and losses (net) on investment transactions, reserved	249,416.67	203,913.38
	531,973.43	482,760.14
Due to special funds	1,822.04	—
	$533,795.47	$482,760.14
Building Fund		
Mortgage (balance of principal; original amount, $200,000.00) on real estate (payable $3,750.00 quarterly for principal and interest, and balance due November 1, 1959)	$152,982.09	$160,874.68
Principal of fund (after deducting $38,588.81 for furniture, fixtures and equipment removed from assets in 1956)	315,419.02	342,815.24
	$468,401.11	$503,689.92
Prize Funds		
Principal of funds:		
George Frederick Kunz Prize Fund	$ 1,344.01	$ 1,293.34
A. Cressy Morrison Prize Fund	18,091.16	17,461.74
	$ 19,435.17	$ 18,755.08
Special Funds		
Principal of funds:		
Grants-in-aid	$ 78,347.60	$ 30,181.19
New York Society of Electron Microscopists	1,153.61	1,017.03
Contributions: Women's Auxiliary	2,362.39	1,492.07
Furniture and fixtures	176.15	176.15
Auditorium project fund	110.82	108.39
Unexpended investment income of endowment funds	4,071.77	2,439.33
	$ 86,222.34	$ 35,414.16

THE NEW YORK ACADEMY OF SCIENCES

Statement of General Fund Income and Expenses and Surplus
Year ended October 31, 1956

Income:		
Membership dues		$170,349.36
Sales of publications		91,739.90
Contributions-in-aid		63,008.55
Contributions – use of building facilities		35,451.38
Contributions to general fund		1,312.00
Income from securities of endowment and building funds		27,243.41
		389,104.60
Expenses:		
Publications costs (including salaries and expenses of printing division)	$106,689.93	
Distribution of publications	16,509.23	
Postage	19,343.91	
Use of building, conferences and meetings expenses	76,578.61	
Building operating salaries and expenses	48,615.65	
Executive salaries and expenses	24,884.13	
Editorial staff – salaries and expenses	24,488.21	
Membership expansion – salaries and expenses	15,595.59	
Public relations – salaries and expenses	4,836.81	
Societies administration	5,381.17	
Social security taxes	2,251.12	
Interest on mortgage	7,048.21	
Accounting salaries and expenses	11,143.23	
General office salaries and expenses	14,971.59	378,337.39
Excess of income over expenses for year		10,767.21
Less:		
Unexpended investment income of endowment funds transferred to special funds		4,071.77
General fund surplus for year		6,695.44
General fund surplus as of October 31, 1955		6,988.26
		13,683.70
Deduct:		
Contribution from general fund to building fund – equivalent to the payments of mortgage principal		7,892.59
General fund surplus as of October 31, 1956		$ 5,791.11

AWARDS

(1) *The A. Cressy Morrison Awards in Natural Science.* After careful consideration and comparison of the eligible papers presented in competition this year, the Committee unanimously made the following awards:

An award of $400 to William Low, Enrico Fermi Institute for Nuclear Studies, University of Chicago, Chicago, Ill., for the paper *The Paramagnetic Resonance and Optical Spectra of Some Ions in Cubic Crystalline Fields.*

An award of $400 to Guy-Laurent Rémillard, Université de Montréal, Montreal, P.Q., Canada, for the paper *Histochemical and Microchemical Observations on the Lipids of the Interscapular Brown Fat of the Female Vespertilionid Bat, Myotis lucifugus lucifugus.*

(2) *The Boris Pregel Award in the Field of Natural Radioactive Substances.* As no entries were received, no awards were made.

(3) *The D. B. Steinman Award for Research in Structural Engineering.* No paper completely fulfilling the conditions for competition was presented to the Academy during 1956. The Committee accordingly made no award in this field.

ELECTION OF FELLOWS AND HONORARY LIFE MEMBERS

The following Members were elected to Fellowship:

Agate, Frederic J., Jr., Ph.D.
Alpert, Norman R., Ph.D.
Alvarez, Walter Clement, M.D.
Ambler, John J., M.D.
Ambrus, Julian L., M.D.
Amos, Dennis Bernard, M.D.
Austrian, Robert, M.D.
Bakwin, Harry, M.D.
Bean, William Bennett, M.D.
Behm, Hans J., B.A.
Bendich, Aaron, Ph.D.
Bennett, Ivan L., Jr., M.D.
Bennett, Leslie R., M.D.
Bentinck-Smith, John, D.V.M.
Berl, Walter George, Ph.D.
Berlin, Nathaniel I., Ph.D.
Bird, Joseph G., Ph.D.
Blank, Harvey, M.D.
Borison, Herbert L., M.S.
Bostick, Warren L., M.D.
Brady, Joseph V., Ph.D.
Brill, Rudolf F., Ph.D.
Brown, Ivan W., Jr., M.D.
Brown, Raymond A., Ph.D.
Buchsbaum, Ralph, Ph.D.
Burdette, Walter J., M.D.
Claff, C. Lloyd, B.A.
Clark, Herbert Mottram, Ph.D.
Coffin, David L., D.V.M.
Colter, John Sparby, Ph.D.
Conklin, Harold C., B.A.
Converse, John Marquis, M.D.
Coombs, R. R. A., Ph.D.
Crosby, William H., M.D.
Curnen, Edward C., Jr., M.D.
Danowski, Thaddeus Stanley, M.D.
Darling, Robert Croly, M.D.
Davis, Dorland J., M.D.
Davis, William W., Ph.D.
Degkwitz, Robert Rudolf, M.D.
De Mello, Lopo, M.D.
Denkewalter, Robert G., Ph.D.
Deterling, Ralph Alden, Jr., M.D.
Dews, Peter Booth, Ch.B.
Dmochowski, Leon, M.D.
Dolman, C. E., Ph.D.
Downs, Cora M., Ph.D.
Eichwald, E. J., M.D.
Elliott, Robert H. E., Jr., M.D.
Enders, John Franklin, D.Sc.
English, James, Jr., Ph.D.
Farr, Lee E., M.D.
Fawcett, Don W., M.D.
Ferris, S. W., B.Ch.
Fine, Jacob, M.D.
Fisher, John C., D.Sc.
Flexner, Louis B., M.D.
Flood, Merrill M., Ph.D.
Furchgott, Robert F., Ph.D.
Ginell, Robert, M.S.
Goldfinger, George, Ph.D.
Goldsmith, Alfred N., D.Sc.
Gordon, Hirsch L., M.D.
Gross, Paul Randolph, Ph.D.
Gruse, William A., Ph.D.
Gulick, John, Ph.D.
Haag, Harvey B., M.D.
Harris, Raymond, M.D.
Hartman, Paul, P.E.
Heden, Carl Goran, M.D.
Hefferline, Ralph Franklin, Ph.D.
Hestrin, Shlomo, Ph.D.
Hoch, Paul H., M.D.
Holaday, Duncan A., M.D.
Hughes, Edward C., M.D.
Hurvich, L.M., Ph.D.
Hutchings, Brian L., Ph.D.
Jacoby, Kurt
Jahn, Theodore L., Ph.D.
Kaliss, Nathan, Ph.D.
Kazal, Louis A., B.S.
Kempf, John Emerson, M.D.
Kennard, Margaret A., M.D.
Keur, Dorothy Louise, Ph.D.
King, John A., Ph.D.
Klein, George, M.D.
Knight, C. Arthur, Jr., Ph.D.
Kolb, Lawrence C., M.D.
Kritchevsky, David, Ph.D.
Krol, Edward J., M.D.
Lasker, Mary, L.H.D.
Leszynski, Werner, Ph.D.
Lewis, Bernard, Ph.D.
Little, C. C., D.Sc.
McBee, Earl T., Ph.D.
McClelland, Laurella, M.D.
McIntyre, A. R., M.D.
Maitland, Hugh Bethune, M.D.
Maren, Thomas H., M.D.
Melnick, Joseph L., Ph.D.
Metraux, Rhoda, Ph.D.
Moe, Gordon K., Ph.D., M.D.
Moore, Alice E., M.D.
Mountain, Isabel Morgan, Ph.D.
Neter, Erwin, M.D.
Nicoll, Paul Andrew, Ph.D.
Oberling, Charles, M.D.
Pait, Charles Franklin, M.D.
Pasternak, Varsenig Z., Ph.D.
Pi-Sunyer, J., M.D.
Pollard, Ernest, Ph.D.
Proskauer, Eric S., Ph.D.
Reese, Thomas Whelan, Ph.D.
Richter, Maurice N., M.D.
Robbins, Frederick C., M.D.
Rogers, Blair Oakley, M.D.
Rogers, David Elliott, M.D.
Rosenberg, Hans R., Sc.D.
Routien, John B., Ph.D.
Salmon, Eleanor Seely, Ph.D.
Samoff, Stanley J., M.D.
Schade, Arthur L., Ph.D.
Schein, Marcel, Ph.D.
Shidlovsky, Boris Alexander, B.A.
Shoben, Edward Joseph, Jr., Ph.D.
Shrigley, Edward White, M.D.

Simeone, Florindo A., M.D.
Smith, Margaret H.D., M.D.
Solmssen, Ulrich V., Ph.D.
Spink, Wesley W., M.D.
Spitz, Rene A., M.D.
Steigman, Alex J., M.D.
Strumia, Max Maurice, M.D.
Stuart-Harris, C. H., M.D.
Surrey, Alexander R., Ph.D.
Sydenstricker, Virgil Preston, M.D.
Syverton, Jerome T., M.D.
Tannenbaum, Jane Belo
von Hippel, A. R.
Wasserman, Louis Robert, M.D.
Waymouth, Charity, Ph.D.
Wiselogle, Frederick Y., Sc.D.
Weiner, Nathan, Ph.D.
Weyer, Edward M., Jr., Ph.D.
Zwolinski, Bruno John, Ph.D.

Honorary Life Memberships were conferred upon the following eminent scientists:

Karl Friedrich Bonhoeffer, Max-Planck Institute of Physical Chemistry, Berlin, Germany.
Vere Gordon Childe, University of Edinburgh, Edinburgh, Scotland.
Charles Alfred Coulson, Oxford University, Oxford, England.
Robert von Heine-Geldern, University of Vienna, Vienna, Austria.
Sir Cyril Norman Hinshelwood, Oxford University, Oxford, England.
James Tutin Irving, Dental Research Unit, Oral and Dental Hospital, Johannesburg, South Africa.
Gregorio Maranon y Posadillo, University of Madrid, Madrid, Spain.
Herbert Harold Read, University of London, London, England.
H. Schaefer, University of Heidelberg, Heidelberg, Germany.
Adolph H. Schultz, University of Zurich, Zurich, Switzerland.
Wilson Smith, University of London, London, England.

ELECTION OF OFFICERS

Report of the Tellers. The tellers appointed by President Root to count the ballots for the election of Officers and other Members of the Council to serve for the year 1957 are pleased to report that the official slate, as nominated by the Council, was elected by a vote of 97 per cent of the 3,682 ballots returned from the Membership.

The list thus chosen by the Membership of the Academy for the year 1957 is as follows: –

President-Elect
BORIS PREGEL

Vice-Presidents
E. J. KEMPF HILARY KOPROWSKI

Recording Secretary
EMERSON DAY

Corresponding Secretary
FREDERICK C. NACHOD

Treasurer
ROBERT F. LIGHT

Councilors (1957-1959)
GEORGE H. MANGUN
MINA REES
HAYDEN C. NICHOLSON
WILLIAM W. WALCOTT

Finance Committee
HARDEN F. TAYLOR, *Chairman*
GORDON Y. BILLARD JOHN TEE-VAN

Chairmen of Sections
M. HALL TAYLOR, *Geology and Mineralogy*
ALBERT S. GORDON, *Biology*
RALPH F. HEFFERLINE, *Psychology*
MARGARET MEAD, *Anthropology*
ROBERT NEILSON BOYD, *Physics and Chemistry*
JEROME SPAR, *Oceanography and Meteorology*
PAUL HARTMAN, *Mathematics and Engineering*

Chairman of Division
M. L. LITTMAN, *Mycology*

PROGRAM OF THE EVENING

After the Business Meeting the following program was given:

THE CASE FOR ANIMAL EXPERIMENTATION*

By Walter S. Root

Retiring President, The New York Academy of Sciences, New York, N. Y.
and
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Animal experimentation is fundamental to all biological work. Too few people realize that almost all new medicines, not just a few, require preliminary tests on animals before they are considered safe for human beings. This lack of information on the part of the public is entirely the fault of the medical scientists who, in the past, have been reluctant to state the case plainly and completely.

The life-saving results of animal experimentation[1,2,3] fill many thousands of volumes. Indeed, a great portion of all medical literature is composed of reports of the discoveries derived from studies on animals. Practically all of our knowledge concerning the functions of the organs of the body, nutrition, the actions of drugs, and surgery has been obtained from such investigations. Without test animals (TABLE 1) we should know little about vitamins or hormones. Within our own time the use of experimental animals has decreased the hazards of smallpox, plague, diphtheria, diabetes, pernicious anemia, typhoid fever, and infantile paralysis. Certainly we can consider a future in which suffering from heart disease, cancer, and tuberculosis will be greatly diminished. Nor have all the benefits accrued to man, for the sciences of animal husbandry and veterinary medicine have developed almost entirely through animal research.[4] Great epizootic diseases such as anthrax, swine fever, chicken cholera, silkworm disease, pleuropneumonia, glanders, Texas cattle fever, black leg, and tuberculosis in cattle, which previously killed millions of animals yearly, have been brought under better control. The health of pets and livestock and the health of human beings are equally dependent upon medical research.

It should be equally obvious that the use of animals is as essential for classroom teaching as it is for medical research. Animals are as necessary in teaching the medical student as "the use of lathes in the training of mechanics, or musical instruments in training musicians, or water in training swimmers."[5] The employment of animals in the classroom enables the student to learn properly to handle delicate living tissue and

*Earlier literature on animal experimentation is cited in *Animal Experimentation and Medical Progress*, by W. W. Keen, Houghton Mifflin Co., Boston, Mass. 1914. Material published since January 1, 1914, may be found in *Bulletin of the Illinois Society for Medical Research*, No. 6, October 1954, Chicago, Ill.

TABLE 1

THE USE OF ANIMALS FOR BIOLOGICAL STANDARDIZATION AND THE ESTABLISHMENT OF STANDARDS OF SAFETY*

Drug	*Test animals*
Diphtheria antitoxin	Horse, rabbit, guinea pig, mouse
Diphtheria toxin	Guinea pig, mouse
Schick test toxin (diphtheria)	Rabbit, guinea pig
Scarlet fever streptococcus	Horse, human, rabbit, guinea pig, mouse
Scarlet fever streptococcus toxin	Human, rabbit
Human plasma	Human, guinea pig, mouse, rabbit
Human immune globulin	Human, guinea pig, mouse, rabbit
Human measles immune serum	Human, guinea pig, mouse, rabbit
Human serum	Human, guinea pig, mouse, rabbit
Human scarlet fever immune serum	Human, guinea pig, mouse, rabbit
Tetanus antitoxin	Horse, guinea pig, mouse, rabbit
Tetanus toxoid	Guinea pig, mouse, rabbit
Antimeningococcic serum	Horse, mouse, guinea pig, rabbit
Antipneumococcic serum	Horse, rabbit, mouse, guinea pig
Typhoid vaccine	Mouse, guinea pig, rabbit
Typhoid and paratyphoid vaccine	Mouse, guinea pig, rabbit
Rabies vaccine	Rabbit, mouse, guinea pig
Smallpox vaccine	Calf, rabbit, guinea pig, mouse
Old tuberculin	Human, guinea pig, mouse, rabbit
Liver extract	Human
Adrenal gland	Dog
Epinephrine ampules 0.1% sol.	Dog
Insulin	Rabbit
Parathyroid extract	Male dog
Posterior pituitary extract	Guinea pig
Stomach powder	Human
Cod liver oil	Rat
Halibut oil capsules	Rat
Vitamin A oil capsules	Rat
Viosterol in oil	Rat
Concentrated vitamin A & D capsules	Rat
Extract of rice bran	Rat
Hexavitamin capsules	Rat
Water for injections	Guinea pig, rabbit
Physiological salt solution	Rabbit, guinea pig
Ringer's solution	Rabbit
Dextrose ampules	Rabbit
Dextrose and sodium chloride ampules	Rabbit
Anticoagulant solution of sodium citrate	Rabbit
Arsphenamine	Rat, rabbit
Neoarsphenamine	Rat, rabbit
Sulfarsphenamine	Rat, rabbit
Tyrpasamide	Rat, rabbit
Ergot fluid extract and extract	Rooster
Digitalis: capsules, leaves, powder, ampules, extract, tablets, tincture	Cat
Ouabain ampules	Frog
Strophanthin ampules	Frog
Strophanthus tincture	Frog
Apocynum fluid extract	Cat
Convallaria	Cat
Aconite fluid extract and tincture	Guinea pig
Penicillin preparations	Rabbit, mouse
Streptomycin	Mouse, rabbit

*As listed in the *United States Pharmacopeia* and the *National Formulary* (Brewer, N., 1954. Animal experimentation. Encyclopedia Britannica, 1: 965).

thus gain a sound understanding of the mechanisms of the living body. Other teaching aids are used extensively, but none completely supplants the need to attain a first-hand knowledge of the living organism.

The use of animals for experimental purposes goes back at least to the time of Galen (born 130 A.D.), and it is the technique employed more and more as the use of the scientific method[6] has been increasingly appreciated. Before the discovery of anesthesia, animal experiments were performed with great difficulty, and the results were hard to interpret. Another important development was the introduction of aseptic methods that have made it possible to subject all tissues and regions of the body to surgical interference.

Medical investigation utilizes animals as diverse as microscopic protozoa and cattle. Most frequently used are mice, rats, chickens, guinea pigs, hamsters, rabbits, cats, dogs, goats, sheep, pigs, and monkeys. The particular animal used depends upon the type of study undertaken. For certain kinds of research the dog is indispensable. Its anatomy and physiology very closely resemble those of man. The organs of smaller animals are usually of insufficient size for teaching or training in surgical skills. The dog also suffers from a number of diseases that afflict man. Chimpanzees and apes can replace dogs in some instances, but they are very expensive and difficult to raise. Moreover, they are vegetarians; unlike those of the dog, their digestive processes are not completely similar to those of human beings.

To summarize, animals are used for experimental purposes in: (1) the acquisition of basic knowledge about bodily functions; (2) the development of new remedies for or preventatives of disease; (3) the teaching and perfecting of knowledge and skills, especially in surgery; (4) the routine testing of medicines as required by law (TABLE 1); and (5) diagnostic tests (TABLE 2).

Unfortunately the use of animals for experimental purposes has not been without opponents. A militant organization was founded in England in 1875, under the name of the British Union for the Abolition of Vivisection. The American Anti-Vivisection Society was organized in 1883.[7] The movement opposed to animal experimentation comprises highly diverse individuals representing all shades of opinion.[8,9,10,11] At one extreme we find members of the lunatic fringe in whom the emotional response produced by the mere mention of animal experimentation is so strong that it is beyond the reach of reason. At the other extreme are those who, while professing great admiration for medical research, would bring about restrictive changes through legislation.[12] Between these groups are many fine, warm-hearted, well-meaning people who are sincerely worried about the obligation that must be assumed for the well-being of animals domesticated or kept in captivity. At present there are in the United States more than a hundred societies opposed to experimentation

with animals, including three national associations. The activities of these groups have taken either one or both of two forms: (1) the infiltration of various humane societies, with their subsequent use for propaganda purposes, and (2) attempts to eliminate or to limit animal experiments by obtaining the enactment of restrictive legislation. By 1945 the opponents of animal experimentation had achieved sufficient strength and influence to sponsor a bill in the New York State Legislature forbidding animal experimentation altogether and to obtain its passage by the state Senate. All work in some key centers of heart, cancer, and atomic energy research was threatened. Fortunately, the bill was killed in the state Assembly.

Either as individuals or acting as a group, anti-vivisectionists have made many violent, false charges of cruelty and torture.[13,14] Many uninformed individuals doubtless are led to believe that, where there is so much smoke, there must be some fire, for most of us erroneously assume that the public enjoys some automatic protection against false propaganda and lying allegations. The facts, however, are to the contrary.[5] The statutes on perjury relate to deliberate lying under oath; the laws of libel, even those provided in the criminal statutes, cover cases

TABLE 2

THE USE OF ANIMALS FOR DIAGNOSTIC PURPOSES*

Condition	*Animal(s) used*
Tuberculosis	Guinea pig, rabbit
Anthrax	Guinea pig
Glanders	Male guinea pig
Listerellosis	Guinea pig, rabbit
Rabies	Mice
Psittacosis	Mice
Equine encephalomyelitis of man	Guinea pig, mice
Foot and mouth disease	Guinea pig, bovine, swine
St. Louis encephalitis	Pigeons, nursing mice
Rift Valley fever	Sheep
Louping ill	Monkeys, mice, sheep
Rat bite fever	Guinea pig, mice
Haverhill fever	Mice, guinea pig
Weil's disease	Guinea pig
Typhus fever	Guinea pig
Rocky Mountain spotted fever	Guinea pig
Tsutsugamushi disease	Mice
Yellow fever	Rhesus monkey, mice
Lymphocytic choriomeningitis	Guinea pig, rats, mice
Poliomyelitis	Monkeys, guinea pigs
Meningococcal infections	Mice
Pneumonia (Pneumococcal infections)	Mice
Tularemia	Guinea pigs, mice
Early pregnancy	Rabbits
Sex hormone concentrations in blood and urine	Rats, mice
Urinary gonadotrophin	Rabbits

*Brewer, N. 1954. Animal experimentation. Encyclopedia Britannica. 1: 965.

in which it can be demonstrated that a particular individual has suffered substantial damage due to slander or libel. It is obviously difficult for the scientist to obtain legal protection against false propaganda.

No matter how phrased, the charges made against those engaged in animal experimentation are almost always accusations of cruelty and the infliction of pain. As Lane-Petter[15] has pointed out, these two elements are separate entities. Pain is inseparable from life itself, and the occasional infliction of pain in our dealings with one another and with our animals must be reckoned among the vicissitudes of living. Cruelty, however, is another matter. Here we are dealing with a disposition to inflict suffering, to take delight in or to exhibit indifference to the pain of another. Behavior of this kind is generally looked upon as a sign of abnormality. There is no reason to regard scientists as more abnormal than other men. Certainly, those whose lives are dedicated to the relief of suffering should not be accused of harboring an opposite objective.

Should we accept the contention that great good can be obtained at the cost of inflicting some pain in a number of animals, we become involved in the old argument that the end justifies the means. Perhaps the soundest conception is that found in the Catholic Encyclopedia:[16] "There is no branch of medical science that cannot be essentially benefited by experiments on animals. In the last instance the results of experiments do good to humanity. Consequently, it appears inadmissable to declare vivisection a means morally forbidden, and to characterize experiments on animals as the torture of animals."

Scientists do not differ from other men in their reaction to ethical and moral considerations. Perhaps for this reason we find certain codes of conduct subscribed to and followed. In the laboratories of The College of Physicians and Surgeons, Columbia University, New York, N.Y., we have posted, as experimenters do in other such laboratories, the following regulations:

Rules Relative to the Use of Experimental Animals

1. All animals used for experiments must be obtained through the Department of Animal Care, or by full agreement with the Department. All animals purchased for experimental use must be obtained from reputable dealers. Stray animals, pets, or discharged war dogs must not be used for experimental purposes.

2. No animals will be provided for any experiment without the approval and written authorization of the Executive Officer of the Department or his deputy, and unless the special care of the animals for the entire period of the experiment has been discussed and arranged with the Department of Animal Care.

3. In experiments involving surgical operations, no operation may be

undertaken without the approval of the Executive Officer of the Department. The Executive Officer will give such approval only after the experimenter has submitted an outline of the plan of the experiment and the details of the kind of animal to be used, operative procedure, and other pertinent information.

4. No surgical operation may be done except in an operating room which is equipped and staffed like an operating room for human patients, so that when survival is contemplated, proper aseptic conditions can be maintained.

5. No surgical operation may be done unless an anesthetic is used.

6. No surgical operation may be done unless provision is made for proper postoperative care. The animal must be kept properly covered in a well-warmed room for at least the first 24 hours after the operation. A special recovery room must be provided for this purpose. Provision must be made during the recovery period for frequent inspection of the animals so that they may receive treatment for discomfort and their well-being assured. Animals must be given the same postoperative care that is customary for human individuals.

7. If, in the course of an experiment, an animal gives evidence of suffering severe or prolonged pain, steps must be taken at once to relieve it, or the animal must be destroyed.

8. The use of animals in teaching and demonstration in student courses must be restricted to the minimum necessary for effective instruction, and the requirements and standards for the care of these animals must be the same as for those used in research.

The maintenance and care of laboratory animals is an expensive undertaking.[17] It has been estimated that properly constructed animal quarters cost up to seven times as much per unit of floor space as a new house. Most institutions provide professional veterinary care. Improvement in the care of laboratory animals has received great impetus from the work of the Animal Care Panel, Chicago, Ill. This organization, founded by N. R. Brewer, has among its members many distinguished medical scientists. The biochemists and the nutritionists have created accurately formulated diets for all types of laboratory animals. Geneticists have produced closely inbred strains whose homozygous genetic constitution results in uniformly dependable reactions to any environmental condition or experimental procedure. The pathologist and epidemiologist have eliminated many of the diseases so commonly endemic among laboratory animals, and they have controlled the epizootic diseases that have so often vitiated the results of many months of painstaking and important research. The zoologists have introduced new experimental animals that have expanded the frontiers of medical research by providing susceptibilities not previously found in commonly available laboratory animals. In its *Proceedings, News Letter,* and other pertinent literature, the

Animal Care Panel provides the means by which the latest and most scientific information on the care and management of laboratory animals can be obtained.

In the United States the regulations covering animal experimentation vary in the different states and even in different cities.[7] The federal Congress can enact regulations of this kind only for the District of Columbia. Bills against animal experimentation have been introduced each biennium since 1897, but none has passed.[18] At present about nineteen states have affirmative laws on animal experimentation; through legislation six states have made definite provisions to supply dogs and cats needed for scientific study from unclaimed pound animals that would otherwise be uselessly destroyed; and thirty-one cities have adopted similar practices.

It is the enactment of pound legislation, controlling the disposal of unclaimed animals, that has thrown the case of animal experimentation into the political arena, thus involving the ordinary citizen. We often assume that the usual humane society is a rest home for aged, unwanted, or lost pets. The truth is that many so-called animal shelters function principally as execution chambers.[25] The figures available for two eastern cities show (TABLE 3) that more than 88,000 dogs and over 200,000 cats were collected annually. Nearly all of the cats and 86.5 per cent of the dogs were destroyed. It is estimated that no less than four million animals are futilely destroyed each year in this country.[7] Despite this, various laboratories carrying out important work on cancer, heart disease, and the hazards of radiation often have difficulty in obtaining experimental animals.

To remedy this situation the New York State Legislature enacted the Metcalf-Hatch Law that permits the New York State Health Commissioner to requisition unwanted stray animals for purposes of medical research. This right of the State Health Commissioner was contested by the Dog Owners Association of New York State and the Westchester County Dog Protective Association, which claimed that the requisitioning of stray dogs from their pounds was in effect a seizure of private property for public use without just compensation; that private property was being taken without due process of law; and that there was an illegal and unconstitutional delegation of power of condemnation to the State Commissioner of Health. The New York State Society for Medical Research, represented by the New York law firm of Webster, Sheffield and Chrystie, which gave its services without recompense, intervened as codefenders with the State Health Commissioner. Supreme Court Justice Elbert T. Gallagher dismissed the claims, holding that animal welfare societies do not have any property right to unlicensed, unwanted, unclaimed animals and are merely custodians for the state. On July 14, 1954, the New York State Court of Appeals affirmed in all respects the decision of the lower tribunal.[19]

TABLE 3

ANIMALS ANNUALLY COLLECTED AND DESTROYED IN TWO EASTERN CITIES*

	Total	*Placed*	*Destroyed‡*	*Per cent destroyed*
New York**				
Dogs	59,552	4,442	55,110	92.5
Cats	155,124	395	154,729	99.7
Philadelphia †				
Dogs	28,936	7,409	21,527	74.1
Cats	47,185	627	46,558	98.7
Total	76,121	8,036	68,085	89.3
Both cities				
Dogs	88,488	11,851	76,537	86.5
Cats	202,309	1,022	201,287	99.5

*Harrison, J. W. E. 1947. Food Drug Cosmetic Law Quart. : 411.
**One reporting agency.
†Three reporting agencies.
‡Includes injured or ill animals.

Recently, the campaign of the opponents of animal experimentation has taken a different direction; now they put the emphasis on "regulation" rather than "abolition."[12] It has been proposed that a law, identical with or similar to the British Act of 1876,[20] be passed in this country. This suggestion refers to the Cruelty of Animals Act of 1876 that regulates animal experimentation in England and some of the British colonies. The Act of 1876 was followed by the Protection of Animals Act of 1911. The latter, however, states, "Nothing in this section shall render legal any act unlawfully done under the Cruelty of Animals Act of 1876."[7] This law creates an elaborate licensing and inspection system; prohibits experiments for the purpose of gaining manual skill; and requires special licenses for teaching demonstrations, for performing experiments without anesthesia, and for keeping an animal alive after a surgical procedure. Laboratories are inspected periodically and the keeping of records is compulsory. The act, which is strictly enforced, requires the expenditure of a large amount of time on paper work.[18]

It should be realized that the Act of 1876 became law in a scientific atmosphere quite different from that which exists today. The British surgeon, Stephen Paget, has pointed out[21] that the science of bacteriology did not then exist, for Pasteur's work was still too recent. In pathology a few experiments were made on tubercle, and a few in surgery, in pharmacology, and on the action of poisons, especially snake venom. The great developments of biochemistry, pharmacology, experimental surgery, experimental pathology, and bacteriology were still in the future. Great credit is due our British colleagues who have labored under the restrictive provisions of the Act of 1876.

In 1906 a British Royal Commission was appointed to investigate the entire subject of vivisection. The commission sat from October of 1906 to March of 1908 and heard 21,761 questions and answers. Attempts on the part of antivivisectionists to misrepresent the nature of the evidence given before the commission led the supporters of animal experimentation in January of 1908 to found the Research Defense Society under the presidency of Lord Cromer. By 1950 the great advances in medical science resulting from animal experimentation led J. Douglas Robertson, the distinguished physician who was then its Honourable Secretary, to change the name of the Society's publication to *Conquest*. The high caliber of the contributions to this magazine has aided greatly in informing the laity of the issues involved in animal experimentation. A similar organization, The National Society for Medical Research, Chicago, Ill., has been founded in this country under the sponsorship of the Association of American Medical Colleges.

One event in connection with the English experience is worthy of note. In 1945 the Court of Appeal rejected the contention that the antivivisectionists were entitled to tax exemption.[22] During the past year the United States Bureau of Internal Revenue filed suit against the American Antivivisection Society to deny tax-exempt status to that organization.

Another country that has regulated animal experimentation is Sweden.[18] Since 1944 it has been necessary to obtain permission to use warm-blooded animals for experimental purposes from the boards of health. Applicants must state the purpose and plan of the experiment, the number and kind of animals to be used, the place where the work is to be done, and the facilities available for the proper care of the subjects. The director of the hospital or institution is held responsible for all such work.

The only nation to prohibit animal experimentation has been Germany under the Nazi government. As a result of this kind of idealism, biological hypotheses were tested upon human beings without their consent in the infamous concentration camps.[23]

It has been stated recently that agitation against vivisection is now at its lowest ebb.[24, 25] Although this may well be true, we must realize that the situation may change at any moment. The treasuries of the societies that oppose animal experimentation have not diminished and the program of these associations are still designed to appeal to the emotions. However, we can find encouragement in the fact that we have received support from the public wherever we have explained our position. Thus, a poll conducted by the National Opinion Research Center of the University of Chicago, Chicago, Ill., under a grant provided by the Rockefeller Foundation, New York, N.Y., showed that 85 per cent of the general population favor the use of live animals in medical teach-

ing and research. Eight per cent were opposed. The results of local referendum votes that have favored animal experimentation are as follows:

Park Ridge, Ill., 1950	4:1	(80 per cent)
Des Plaines, Ill., 1950	2¾:1	(73 per cent)
Los Angeles, Calif., 1950	1¾:1	(64 per cent)
Baltimore, Md., 1950	4⅓:1	(81 per cent)

The differences between the percentages shown in the referendum votes and those found in the opinion poll must reflect the effectiveness of the propaganda used by the antivivisectionists.[26] In spite of the distractions and annoyances caused by irresponsible claims and charges, we can face the future, assured of the service rendered by animal experimentation and confident of the understanding of our fellow citizens.

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Note. A paper entitled "The International Geophysical Year" was presented at the Annual Dinner and Meeting of the Academy on December 6, 1956, by Joseph Kaplan, Professor of Physics, University of California at Los Angeles, Los Angeles, Calif., and Chairman, United States National Committee for the International Geophysical Year, Washington, D. C. This paper will appear in the February issue of the *Transactions*.

ABSTRACTS OF WINNING PAPERS

THE PARAMAGNETIC RESONANCE AND OPTICAL SPECTRA OF SOME IONS IN CUBIC CRYSTALLINE FIELDS

By William Low*

Accorded an A. Cressy Morrison Award in Natural Science, 1956

It is shown that paramagnetic resonance spectra can give significant information about the local crystalline field strength and symmetry acting on the paramagnetic ion. This is illustrated on a number of iron group and rare earth group ions in a cubic field. The combined optical and paramagnetic resonance spectra give strong indication that most of the iron group ions are exposed to strong crystalline field in MgO and that considerable covalent bonding occurs. Of particular significance are the spectra of Cr^{3+}, of Fe^{3+} in MgO, and of Gd^{3+} in CaF_2. These spectra are cubic despite the fact that these ions have an excess of charge. Apparently these ions and their surroundings must be regarded as a tightly knit unit very similar to a molecule.

HISTOCHEMICAL AND MICROCHEMICAL OBSERVATIONS ON THE LIPIDS OF THE INTERSCAPULAR BROWN FAT OF THE FEMALE VESPERTILIONID BAT, *MYOTIS LUCIFUGUS LUCIFUGUS*

By Guy-Laurent Rémillard**

Accorded an A. Cressy Morrison Award in Natural Science, 1956

The lipid content of the interscapular brown adipose tissue of *Myotis lucifugus lucifugus* was studied throughout an annual cycle by means of both histochemical and microchemical procedures. In addition, the lipid content was correlated with changes in the fresh and dry weights of the brown adipose tissue, for which purpose the water content and the total solids were likewise determined.

During the summer months, the total weight and the water content of the brown fat tissue fluctuate. Microchemical determinations show that the concentrations of total lipids, total cholesterol, and total fatty acids are relatively high at this time. Histochemical analysis reveals that the cytoplasm of each brown fat cell is filled with droplets of a purely lipid composition.

As the animal approaches the hibernating period, there is a gradual increase in the total weight of the interscapular lobes and in the size of the individual brown fat cells. This is accompanied by an increase in the lipid content of the interscapular tissue.

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During hibernation, on the other hand, there is a gradual decrease in the total weight of the interscapular lobes, and in the amount of water, total lipids, total cholesterol, and total fatty acids. Histochemical analysis, on the other hand, suggests that during hibernation there is a gradual depletion of neutral lipids in the interscapular lobes of brown adipose tissue.

Toward the end of the period of hibernation, the total weight and the water content of the interscapular lobes reach their lowest levels. However, there is a relative increase in the lipid concentration of the brown adipose tissue at this time.

At all seasons of the year the cytoplasm of each brown fat cell contains numerous sudanophilic lipid droplets. The remainder of the cytoplasm of the multilocular adipose cell is filled with granules that apparently do not vary in composition, number, and size throughout the year. Comparisons between sections treated for the demonstration of mitochondria and sections stained by B. L. Baker's acid hematein test suggest strongly that the cytoplasmic granules of the brown fat cell are mitochondria that are especially rich in phospholipids.

THE NEW YORK ACADEMY OF SCIENCES

announces

THREE SCIENTIFIC AWARDS FOR 1957

The New York Academy of Sciences announces the following three awards for the coming year:

I. THE A. CRESSY MORRISON AWARDS IN NATURAL SCIENCE

Two awards of $400 each, to be known as THE A. CRESSY MORRISON AWARDS IN NATURAL SCIENCE, will be given at the Annual Meeting of The New York Academy of Sciences in December 1957 for the two most acceptable papers in a field of science covered by the Academy or an Affiliated Society.

II. THE BORIS PREGEL AWARD IN THE FIELD OF NATURAL RADIOACTIVE SUBSTANCES

Through the generosity of Dr. Boris Pregel, an award of $500 is offered for the most acceptable paper embodying the results of research in the field of natural radioactive substances received during the year 1957. The according of this award will be announced at the Annual Meeting of the Academy in December 1957.

III. THE D. B. STEINMAN AWARD FOR RESEARCH IN STRUCTURAL ENGINEERING

Through the generosity of Dr. D. B. Steinman, an award of $500 is offered for the most acceptable original paper, submitted from the Membership of the Academy, in the field of pure or applied research in structural engineering, or the related science of aerodynamics, or as applied to the analysis and design of civil engineering structures. The purpose is to encourage research development and writing in the subject of engineering sciences. The according of this award will be announced at the Annual Meeting of the Academy in December 1957.

CONDITIONS GOVERNING THESE AWARDS

(1) *Eligibility.* Authors and co-authors applying for awards I and III shall be members in good standing of The New York Academy of Sciences or one of its Affiliated Societies, but nonmembers may become eligible by joining one of these organizations before the closing date.

All scientists engaging in research in the field of natural radioactive substances may make application for award II.

(2) *Date.* Papers are to be submitted on or prior to October 15, 1957, to the Executive Director of The New York Academy of Sciences, 2 East Sixty-third Street, New York 21, New York.

(3) *Papers.* All papers submitted must embody the results of original research not previously published. The manuscript shall be typewritten, in English, accompanied by all necessary photographs, drawings, diagrams, and tables, and shall be ready for publication. Papers must be accompanied by a summary of the data presented and conclusions reached.

(4) *Awards.* The awards shall be made by the Council of The New York Academy of Sciences, and its decision shall be final.

(5) *Publication.* The Academy shall have first option on the publication of all papers submitted, unless especially arranged for beforehand with the authors, but such publication is not binding on the Academy.

(6) Wherever and whenever published, the papers receiving the awards shall be accompanied by one of the three following statements:

In award I, "Accorded an A. Cressy Morrison Award in Natural Science in 1957 by The New York Academy of Sciences";

In award II, "Accorded a Boris Pregel Award for Research on Natural Radioactive Substances in 1957 by The New York Academy of Sciences";

In award III, "Accorded a D. B. Steinman Award for Research in Structural Engineering in 1957 by The New York Academy of Sciences."

Such a statement, in substance, must also accompany any formal publicity initiated by the author regarding the award paper. If published elsewhere with the consent of The New York Academy of Sciences, six copies of each award paper must be deposited shortly after publication with the office of The New York Academy of Sciences.

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Executive Director

SECTION OF BIOLOGY
and
*DIVISION OF MYCOLOGY**

VIRUSES: THE VERSATILE PARASITES

I. THE ARTHROPOD-BORNE GROUP OF ANIMAL VIRUSES**

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The distinguishing property of the arthropod-borne (Arbor) animal viruses is that in nature they multiply in the bodies of arthropods without occasioning detectable damage to their tissues or causing ill effects or death in these sectors. Transmission of these agents to man or other hosts – mammals or birds – takes place through an arthropod bite; the carrier, in turn, becomes infected by ingestion of blood from a host at the time when virus is present in the latter's peripheral circulation.

In the laboratory, all known Arbor viruses are pathogenic for Swiss mice by intracerebral inoculation. This pathogenicity may be present at the initial isolation or may develop later after passing through an experimental host. In fact, the Swiss mouse, either mature or suckling, is the animal of choice for the group as a whole.

The present article concerns: (1) further developments on classification of Arbor viruses, and (2) some of the consequences, both in nature and in experimental animals, that derive from the existence of groups.

Antigenic relationships among these viruses have been the subject of study for some time. Thus, among other instances, by means of neutralization tests Smithburn[1] showed the presence of cross reactions among Japanese B, St. Louis, and West Nile viruses. Casals[2] confirmed this by complement-fixation tests and demonstrated, in addition, that louping-ill and Russian spring-summer (Russ. SS) viruses were closely related to one another. Havens *et al.*[3] indicated some cross reactions between Eastern and Western equine encephalomyelitis (EEE, WEE) viruses; Sabin[4] demonstrated that overlaps existed between the arthropod-borne encephalitides[5] and the agents of dengue and yellow fever. Finally, through the application of methods better suited to the investigation of these relationships, Casals and Brown[6] first studied the subject systematically and then formulated two sharply defined groups, designated as A and B.

*The Section of Geology and Mineralogy held a meeting on December 3, 1956, at which Helgi Johnson, Department of Geology, Rutgers University, New Brunswick, N. J., delivered a paper entitled "Geologic Engineering Problems at Thule Air Force Base, Greenland". This paper will appear in a later edition of the *Transactions*.

The Section of Physics and Chemistry held a meeting on December 4, 1956, at which a paper entitled "Solution Behavior of Blood Plasma Expanders" was presented by Frederick R. Eirich, Department of Chemistry, Polytechnic Institute of Brooklyn, Brooklyn, N. Y. No copy of this paper has thus been received.

**This article, illustrated with lantern slides, was the first of three papers presented at a joint meeting of the Section of Biology and the Division of Mycology on December 10, 1956.

Classification of Arbor Viruses

The present investigation was based on the study of 47 distinct Arbor viruses. These are listed in TABLE 1. Also studied, but not included here, were 5 agents belonging to the new Group C and 8 other agents that seem not to belong in any of the established groups; all of these require further testing. An additional 14 isolates, considered at one time as possible new agents, were investigated and found to be merely new isolates of already established viruses.

Because most of them are well known, no attempt will be made to describe, even briefly, the viruses named in TABLE 1. Of the unfamiliar ones, Chikungunya, Mayaro, and California salivary gland bat virus (Cal. Bat) have been, or are in the process of being, reported by the investigators responsible for their isolation. The remaining viruses are designated by laboratory numbers, and their origin and the investigators that supplied them are as follows: SAT Ar 94 (South Africa, Tongaland, arthropod), SA H 177 (human), SA H 336, and SAT Ar 53 by K. C. Smithburn; Be An 15 (Belem, animal), Be An 17, Be An 848, Be An 73, Be An 277, and Be H 151 (human) by Ottis Causey; Eg Ar 492 (Egypt, arthropod), Eg Ar 1095, and Eg Ar 1152 by R. M. Taylor; Len 731-19 (California) by E. H. Lennette; and Tr 3587 (Trinidad, B.W.I.), Tr 8900, and Tr 9760 by W. G. Downs and C. R. Anderson.

Methods of Study

The grouping as described here is based exclusively on the detection of immunological cross reactions. To this end, three methods were employed; complement fixation (CF), hemagglutination inhibition (HI), and neutralization (NT) tests, either by intracerebral or intraperitoneal routes of inoculation.

The grouping of the Arbor viruses has two aspects. In trying to determine whether a serological overlap exists between two different agents, it is logical to use the method that shows the widest range and the highest titer of cross reaction. Following this, however, another more specific method must be used. Accumulated experience indicates that the tests mentioned can be graded in respect to inclusiveness, from greater to less, HI, CF, and NT by intracerebral route.

The routine procedure for classifying an Arbor virus consisted, therefore, of (1) applying the HI test for general diagnosis of its group, and (2) on the assumption that the HI test had failed to reveal its finer distinction within the group, by applying the CF or intracerebral NT tests. When the virus failed as an agglutinating antigen, the other tests were still used and, on occasion, recourse was had to its hyperimmune serum employed against known hemagglutinating antigens. The intracerebral NT test, though in general more specific, was not used extensively. The intraperitoneal NT test had a greater range of overlapping reactions

TABLE 1

CLASSIFICATION OF ARTHROPOD-BORNE ANIMAL VIRUSES

Group A	*Group B*		*Group C*	*Ungrouped*	
Chikungunya	California bat	SA H 177	Be An 15	Anopheles A	Eg Ar 1152
Eastern EE*	Dengue, type 1	SA H 336	Be An 17	Be An 73	Len 731-19
Mayaro	Dengue, type 2	St. Louis	Be An 848	Be An 277**	Rift Valley fever†
Semliki Forest	Ilhéus	Uganda S		Be H 151**	Sandfly fever, Naples†
Sindbis	Japanese B	West Nile		Bunyamwera†	Sandfly fever, Sicilian†
Venezuelan EE*	Louping ill	Yellow fever		Bwamba‡	SAT Ar 53§
Western EE*	Murray Valley E.	Zika		California, Hammon-Reeves	Tr 3587
	Ntaya			Colorado Tick fever	Tr 8900
	Russian SS			Eg Ar 492	Tr 9760§
	SAT Ar. 94			Eg Ar 1095	Wyeomyia

All viruses in Groups A, B, and C produce hemagglutinins; louping ill not tested.

*EE = equine encephalomyelitis.

**Be An 277 and Be H 151, although not identical, are related by CF.

†These viruses produce hemagglutinins.

‡There seem to be several agents, isolated in South Africa, that are very closely related to Bwamba, but are not identical with it.

§SAT Ar 53 and Tr 9760, distantly related by CF.

within the group, even broader on occasions than that shown by the HI test; this may be correlated to the higher sensitivity of the test.[7] Hence, it was generally noted that whenever a cross reaction was detected by the intracerebral NT test, it could also be shown to exist by CF and, similarly, cross reactions by CF were also apparent in the HI test.

Since HI was found to be the more inclusive test for detection of serological overlaps in the Arbor viruses, *the present grouping is based fundamentally on the behavior of these agents and their immune sera in the HI test.*

Specific details on the preparation of antigens and the procedure of the tests have been reported elsewhere.[6, 8, 9, 10, and 11]

TABLE 2

GROUP A: HEMAGGLUTINATION INHIBITION WITH HYPERIMMUNE MOUSE SERA

Serum	Antigen						
	Chik.	*May.*	*Sem.*	*Sind.*	*WEE*	*EEE*	*VEE*
Chikungunya	8+	4	4	0	3	–	–
Mayaro	7	8+	8+	3	6	3	3
Semliki	3	6	9	1	5	3	4
Sindbis	1	3	3	8	9	4	1
WEE	3	4	3	5	11	4	5
EEE	3	2	2	2	5	11	4
VEE	4	4	3	2	4	5	7+

In this and subsequent tables, the HI titer of a serum is expressed by a number that represents the highest serial twofold dilution of serum that gave complete inhibition of hemagglutination; the first dilution being 1:10 = 1. This number is, in effect, the exponent of the power of two, that, when multiplied by 5, gives the dilution. Thus, a titer represented by 8 = dilution $1:1280 = \frac{1}{5 \times 2^8}$.

The Groups of Arbor Viruses

TABLE 1 shows the present grouping of Arbor viruses and TABLES 2, 3, and 4, illustrate the results of HI tests that have constituted the basic criteria for such distinctions. The work summarized in these four tables follows.

Hyperimmune sera were prepared in mice for each of the 47 viruses listed in TABLE 1; a given sample of each of the sera was tested against all or as many of the 29 hemagglutinating antigens as were available with the exception of Rift Valley fever and louping ill. Each serum was tested against 8 units of antigen in increasing twofold dilutions beginning at 1:10. In order to simplify the tables, the titers of sera are expressed by the ordinal of the highest dilution of serum that gave complete inhibition of hemagglutination; thus, dilutions 1:10, 1:20, 1:40,

TABLE 3

GROUP B: HEMAGGLUTINATION INHIBITION WITH HYPERIMMUNE MOUSE SERA

Serum	Antigen														
	Cal. bat	Den. 1	Den. 2	Ilh.	Jap. B	M.V.E.	Nta.	Russ. SS	SA H 177	SAT Ar 94	St. Lou.	Ug. S	West Nile	Y. fever	Zika
California bat	8				6						7				
Dengue, type 1	5	6	6	5	4		6	5	3	3	5	4	5	4	5
Dengue, type 2	6	3	6	2	1	6	2	4	5	4	3	4	3	2	4
Ilhéus	6	7	6	8	8		7	6	5	5	7	7	8	4	6
Japanese B	6	6	7	7	7	6	7	7	5	4	7	4	6	5	7
Murray Valley E.		4		5	5	8		4			5			3	
Ntaya		5	4	3	3	5	4	4			4	4	4	5	3
Russian SS		3	5	6	5	7	4	7	4	4	6	4	3	5	5
SA H 177			4	5							5			3	
SAT Ar 94															
St. Louis	7	7	7	8	8	9	5	6	6	6	9	7	8	5	7
Uganda S	7	5	7	6	5	6		5	6	6	6	5		6	5
West Nile (Egypt)	7		5	5	6	6	6	5	6	5	6	6	7	3	5
Yellow fever	5	3	2	5	6	3		2	3	0	6	4	4	6	3
Zika			4			7		4	5	5					6

etc. become 1, 2, 3, etc. in the tables. Of a possible total of 1,363 combinations, only about 750 were done with the individual samples of sera listed in TABLES 2, 3, and 4; these 750 combinations do not include repeat or isolated partial trials. Even though not every conceivable combination of serum and antigen was tested, it is reasonable to assume, in view of the total work performed, that the classification arrived at is well grounded. Group A, TABLE 2, comprises at present seven distinct agents; none of the sera reacted with any of the antigens of the other groups. Nor have any sera against viruses not of the group reacted against any of the Group A antigens. Similarly, Group B consists of 17 different agents; TABLE 3 shows results of HI tests with 15. As in Group A, no cross reactions were observed with antigens or sera derived from viruses outside the group. TABLE 4 depicts the situation in a third group, C. The evidence available indicates that at least three different viruses exist in this group, that is, Be An 15, Be An 848, and Be An 17; it is possible that others may be found to belong here.

TABLE 4

GROUP C: HEMAGGLUTINATION INHIBITION WITH HYPERIMMUNE MOUSE SERA

Serum	*Antigen*							
	Be An 15	*Be H 176*	*Be An 974*	*Be An 848*	*Be An 748*	*Be An 3994*	*Be An 947*	*Be An 17*
Be An 15	9	9	7	4		4		1
Be H 176	9	9	8	7		7		2
Be An 974								
Be An 848	0	0	1	7		5		0
Be An 748	4	4	6	10		9		2
Be An 3394								
Be An 947	1	1	3	9		7		0
Be An 17	1	1	2	2		2		7

From the perusal of TABLES 2, 3, and 4, it becomes apparent that, with the systems employed, the HI test revealed in both Groups A and C a degree of specificity sufficient, in general, to distinguish between the homologous and the heterologous reactions. An even greater degree of specificity, amounting often to absence of cross reaction, was found with human convalescent sera, or with sera from animals that had received 1 injection of virus instead of the 5 or 6 given for the production of hyperimmune sera.

By way of contrast, not even such limited specificity was encountered in Group B. There, in general, the homologous and heterologous reactions with hyperimmune sera were similar in magnitude. With simple immune

or with convalescent sera, the homologous reactions were generally somewhat stronger than the heterologous; cross reactions, however, were the rule.

Evidence of cross reactions among the ungrouped viruses (TABLE 1) has been found by CF with some of the agents*; the possibility of identifying additional groups is strong, but sufficient evidence for this is not as yet available.

From results of the less inclusive tests, CF and NT, it has been possible to infer that within both Groups A and B certain viruses have closer connections with one another than with the remaining agents in the group. In Group A, Chikungunya, Mayaro, and Semliki viruses constitute one such tribe; Sindbis and WEE constitute another. In Group B, Ilhéus, Japanese B, Murray Valley Encephalitis, St. Louis, West Nile, and possibly Ntaya viruses seem to constitute a subgroup; dengue 1 and 2, another; and, finally, SA H 336, Uganda S, and yellow fever appear more closely related to each other than to the remaining viruses of Group B. It was already known that Russian SS and louping-ill viruses were closely related.[2]

Significance of Group Relationships

Sharply delimited groups among the Arbor viruses, determined by serological methods, were thus established; investigations were then conducted to find out what influence, if any, these group relationships had on the course of natural or experimental exposure to Arbor viruses, and especially whether group interaction arises in a host that might have been naturally or artificially exposed to more than one virus from a group. The following description covers five selected observations deriving from experimental as well as field studies that bear on the problem. The first three topics deal with materials from experimental animals; the last two cover materials from the field.

1. *Influence of time factor on specificity of HI tests.* As was shown earlier, the results of HI tests with hyperimmune mouse sera indicated that, in general, cross reactions were more pronounced in Group B than in either A or C. With either human convalescent sera or sera from animals given a single injection of virus, Groups A and C as a rule exhibited almost complete specificity while, in Group B, marked overlaps often occurred. It is probable that, at least in some instances with human sera, as will be mentioned later, the time elapsed between inoculation of a virus and collection of the serum to be tested was important in determining the range or pattern of reaction.

Since Group B was the one with most cross reactions, viruses of this group were selected. In one type of experiment, groups of mice were injected subcutaneously once and bled 7, 12, and 18 days later. The

*NT tests have also shown certain cross reactions, not described in this paper (L. Whitman, personal communication).

viruses used were of the St. Louis and Japanese B strains. The results of HI tests carried out with these sera against antigens derived from several Group B viruses are illustrated in FIGURE 1, which shows that 7 days following inoculation with either Japanese B or St. Louis virus, HI antibodies were present to a titer of 6 dilutions (1:320) for each homologous antigen, while the cross-reacting antibody was present to a titer of 2 dilutions (1:20) with some antigens in the Japanese virus-immune mice, and it was almost absent in the St. Louis virus-immune mice. Determinations at 12 and 18 days showed a sharply contrasting picture: development of group antibodies proceeded then at a rate faster than that of the specific ones, with the result that the earlier specificity at 7 days was soon replaced by a marked group reaction.

2. *Synergistic group response.* On the assumption that group relationships are based on antigenic structures common to all the members of a group, it was considered likely that the response to immunization or exposure to one of the viruses of a group would be modified by previous exposure to another member of the same group. Development of HI antibodies with Group A, rather than B, viruses was selected for the experimental approach to this question. The reason for this choice of Group A lay in the high degree of specificity prevailing in it, even in tests after prolonged periods following immunization with any of the viruses. It was also assumed that, under these conditions, it would not be dif-

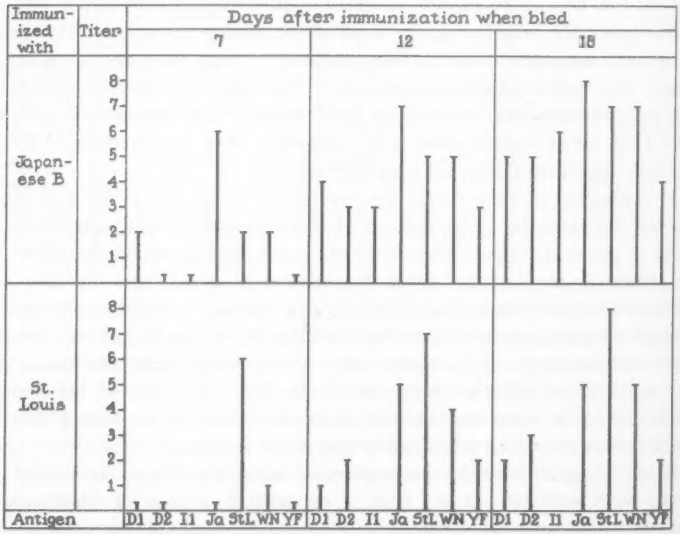

FIGURE 1. Group B. Rates of development of specific and group HI antibodies in mice. For abbreviations see TABLES 6 and 7.

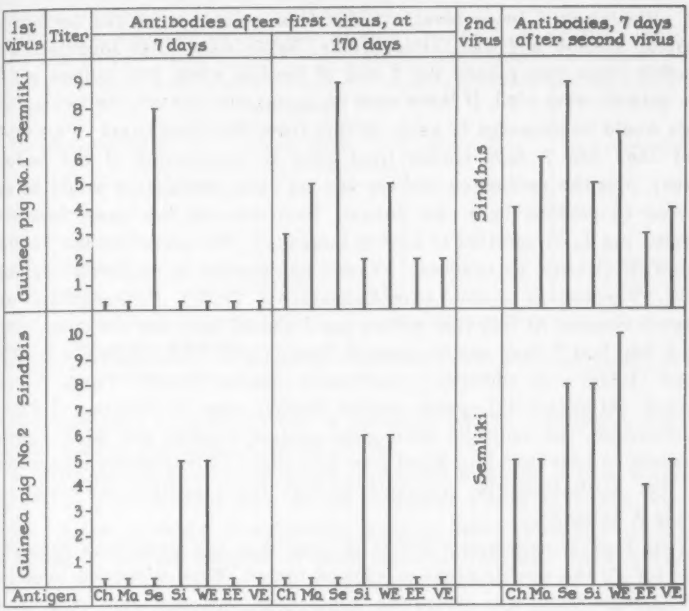

FIGURE 2. Group A. Synergistic action of two different viruses on the development of group antibodies. Ch = Chikungunya. Ma = Mayaro. Se = Semliki. Si = Sindbis. WE = Western equine encephalomyelitis. EE = Eastern equine encephalomyelitis. VE = Venezuelan equine encephalomyelitis.

ficult to detect the development of cross-reacting antibodies following an injection of a second virus.

Guinea pigs were given by intracerebral route a virus (Sindbis, Semliki, or Mayaro), followed by a second intracerebral injection of the same virus or of one of the other two. Samples of serum were taken at intervals and tested against all seven Group A antigens. FIGURE 2 gives the results obtained with two illustrative examples. Guinea pig 1 was given first Semliki virus and, 6 months later, it was given Sindbis; guinea pig 2 was given Sindbis first and, 6 months later, Semliki. In the figure are shown the results of HI tests with the samples of serum. Seven days after the first injection, guinea pig 1 showed a specific response: its serum reacted with Semliki only. Guinea pig 2 exhibited the typical response that has generally followed inoculation with Sindbis: antibodies developed against Sindbis and WEE viruses to the same titer, with no reaction against the remaining antigens. Six months later, immediately before inoculation of the second virus, guinea pig 1 showed in its serum antibodies against Semliki to a titer similar to that which it had had before; but, in addition, group antibodies were now present (cf. Section

1), although at a lower level. At this time guinea pig 2 had antibodies against Sindbis and WEE viruses only. Seven days after inoculation of Sindbis virus into guinea pig 1 and of Semliki virus into guinea pig 2, the animals were bled. If there were no synergistic action, the two guinea pigs would be expected to have, at this time, the same titers of antibody that they had 7 days earlier (just prior to inoculation of the second virus), plus the antibodies that the second virus inoculation would be expected to produce in a new animal. This was not the case, however. Guinea pig 1, in addition to having increased antibodies against Sindbis and WEE viruses, as expected, showed an increase of antibodies against EEE, Venezuelan equine encephalomyelitis (VEE), Chikungunya, and Mayaro viruses. At this time guinea pig 2 should have had the same titers as it had had 7 days earlier against Sindbis and WEE viruses – 6 dilutions (1:320) – in addition to antibodies against Semliki virus. As expected, antibodies did appear against Semliki virus, 8 dilutions (1:1280). Furthermore, not only did antibodies against Sindbis and WEE viruses increase in titer from 6 to 8 and from 6 to 10 (1:5120) dilutions respectively, but antibodies also appeared in high titers against all remaining Group A antigens.

This type of synergistic action of more than one virus was generally true for all the combinations of viruses tested. When a second injection of the same virus was given, the homologous response was, in general, more marked than the cross-reacting one, but the group response was also present.

3. *Influence of group relationship on resistance to infection.* It was already known that repeated inoculations of a virus from Group B resulted in protection ot animals against another virus of the same group, particularly when the challenge was given by peripheral injection and the two viruses were closely allied to each other.[2, 12, 13] Similarly, it was possible to show that a Group A virus, even when inoculated only twice and in small volumes, was capable of protecting mice against a subsequent challenge with another virus of the same group.

In order to explore this problem further and to discover whether such cross reactions may actually be important in the establishing of group immunity in nature, the following experiments were undertaken.

Two lots of mice were immunized: (1) with Chikungunya virus, by subcutaneous inoculation of 0.05 ml. of dilution 10^{-1} of virus; one injection was given on the first day of the test and a second one on the 15th day; (2) with St. Louis virus in the same way. A third control group was uninoculated. Two weeks after the second injection of virus, all mice were challenged by intraperitoneal (IPER) inoculation of 0.2 ml. of Semliki virus in dilutions as indicated in TABLE 5. It can be seen clearly that previous exposure to Chikungunya virus of Group A resulted in an appreciable resistance to challenge by Semliki virus of this group. Im-

TABLE 5

GROUP A: CROSS-PROTECTION AGAINST INFECTION OF MICE WITH SEMLIKI FOREST VIRUS INDUCED BY CHIKUNGUNYA VIRUS

Treatment of Mice	*Challenge Dilution of Semliki Virus, IPER*					*Log L.D.$_{50}$*	*Log protection*
	−4	−5	−6	−7	−8		
None (controls)	6*	7	7	7	3	7.4	
Chikungunya	4	2	1	3	0	4.6	2.8
St. Louis	7	8	7	6	3	7.4	0.0

*Six of 8 mice challenged died.
Log L.D.$_{50}$ = Logarithm of the reciprocal of the highest dilution of virus causing 50 per cent mortality.

munization with St. Louis virus of Group B, on the other hand, did not protect against the Group A Semliki virus.

4. *Influence of group relationship on natural infection in man; antibody pattern in convalescent sera.* In the course of surveying human populations for the presence of antibodies against Arbor viruses, particularly of Group B, certain patterns of group antibody response were revealed, not only by the HI test and to a slightly lesser degree by CF, but also by NT tests. The patterns differed essentially in the degree of specific reactions (that is, freedom from overlapping) within the group.

Attempts were made to secure information on this question in the light of the experimental work reported in Section 2. To this end sera were selected from a large number of individuals who had recently been ill with an infection from a Group B virus. In so far as the available quantity of serum permitted, the sera were tested by CF, HI, and NT tests. Two different types of serological responses to the current infection became evident. In one, the results of NT and CF tests were wholly or almost wholly specific; even the HI test showed, on occasion, a definite degree of specificity for the virus that presumably brought about the infection. In the second type of response, not only the HI and, to a slightly lesser degree, the CF, but even the NT tests indicated a marked group response which, in many instances, could effectively prevent a definite diagnosis unless the virus had been isolated from the individual. The first type of response, more specific, is now postulated to have been associated with infection in a person having no previous contact with another virus of the group; this is designated "single infection." The second type of response, it is now believed, is associated with recent infection in a person who at some time in his past life had been infected with a virus of the same group; this is designated "double infection." A few selected examples are presented in TABLES 6 and 7. In the four cases reported, Japanese B virus infection (TABLE 6) was originally diagnosed by CF;

TABLE 6

GROUP B: ANTIBODY RESPONSE IN MAN FOLLOWING A SINGLE OR A DOUBLE INFECTION

Current infection	Case No.	Serum, days from onset	Antibodies and Antigens																Single or double infection
			HI							CF							NT			
			C.B.	D2	I1	Ja	StL	WN	YF	C.B.	D2	I1	Ja	StL	WN	YF	D2	I1	StL	
Japanese B	1	30		2	2	7	3	3	1		0	0	64	4	4	0				S
	2	30		2	1	7	4	4	0		0	0	64	0	0	0				S
	3	30		7	9	9	9	9	8		8	64	128+	128	32	0				D
	4	30		8	9	8	9	9	6		4	64	128+	64	32	0				D
St. Louis	5	7	0	0	0		4							0						S
		19	1	1	2		5							16						
		180	0	0	2	1	3	2	0					0						
		700	1	1	3	3	4	3	2					0						
	6	20	8	8	9		8													D
		210	8	8	8		8			8	16	32		16			3.4+		2.4+	
		360	8	8	7	8	8	8	7	4	32	32		32			3.4+			
		1050	8	8	8	8	8	8	7	8	32	32		32			3.4+	1.7		

Presumptive diagnosis established by CF test.
HI titers, expressed by the number of tubes showing inhibition.
CF titers, expressed as the denominator of the serum dilution.
NT, expressed as the log difference between titer of virus in control and serum.
C.B. = California bat. D2 = Dengue, type 2. I1 = Ilheus. Ja = Japanese B. StL = St. Louis. WN = West Nile. YF = yellow fever.

TABLE 7

GROUP B: ANTIBODY RESPONSE IN MAN FOLLOWING A SINGLE OR A DOUBLE INFECTION

Current infection	Case No.	Serum, days from onset	Antibodies and Antigens														Single or double infection
			HI						CF				NT				
			D1	D2	I1	StL	WN	YF	D1	I1	StL	YF	D1	D2	I1	YF	
Yellow fever	1	2		0	0	0		0	0	0	0	0		0	0	0	S
		46	0		4		6	7	0	0	0	16	1	0	0	6	
	2	2	0	0	0	0		0	0	0	0	0		0	0	2	S
		46	0		4		6	7	0	0	0	32	1	0	0	6	
	3	5	5		7	7	7	5		16	16	0	6		6	0	D
		35	9		10+	11	11	10+		128+	128+	32	6		6	6	
	4	2	0		0		0	0	0	0	0	0	1	0	6	0	D
		35	5		3		9	7	0	16	8	16	1	3	6	6	
	5	2	0		4		4	2	0	4	0	0	0	0	6	0	D
		32	8		10+		10+	9	8	128+	128+	8	6	2	6	6	

Diagnosis established by isolation of virus.
Result of neutralization tests, expressed as the number of mice that survived in groups of 6, inoculated with undiluted serum and 50-100 $L.D_{50}$ of virus.
D1 = Dengue, type 1.
Remaining footnotes as on TABLE 6.

at the time of this outbreak, the virus was also isolated from mosquitoes trapped in the area. Cases 1 and 2 showed a definitely higher titer by HI test against Japanese B antigen than against the remaining Group B virus antigens. By CF, using 4 to 8 units of antigen, the reaction was either specific or showed only the smallest degree of cross reaction; the cases were considered, therefore, to be single infections by Japanese B virus. Cases 3 and 4, on the other hand, showed absolutely no specificity by HI test and marked overlap by CF; these persons were regarded as cases of double infection, the current one presumably by Japanese B, the older one also by a Group B virus.

The next two individuals, Cases 5 and 6, were diagnosed by CF in another laboratory as St. Louis virus infections. Serial bleedings were available from each. Case 5 by HI test showed an early specific response for St. Louis virus, which, in time, was followed by increasing cross reactions (cf., above, Section 1); but even in the later specimens the reaction with St. Louis virus was still highest. Case 6, on the other hand, showed by HI test a completely nonspecific reaction and at high titers; similarly, the CF test gave a group response. The NT test showed antibodies not only against St. Louis virus, but also against Ilhéus, and particularly against dengue, type 2. It was assumed, therefore, that Case 5 was an example of single infection with St. Louis virus, and that Case 6 was one of double infection, with the possibility, based on the NT results, that the old infection was due to dengue virus; no conclusion could be reached as to the identity of the second infection.

TABLE 7 shows results of tests with sera from individuals infected with yellow fever virus. In each instance, yellow fever virus was isolated from the acute-phase serum. Patients 1 and 2 showed no detectable antibodies in their acute-phase serum but, in the convalescent samples, the titer of antibodies against yellow fever by HI test was higher than any of the remaining titers. The reaction was specific by CF, and the sera protected only against yellow fever by NT test. These were considered single infections with yellow fever virus.

In contrast, Cases 3, 4, and 5 showed a marked group response by HI and CF tests; Nos. 3 and 5 showed this even in the acute-phase serum. In the latter, the result of the NT test was more informative: the serum from each person changed from negative to positive protection against yellow fever virus. Neutralizing antibodies against other Group B viruses were also present, not only in the convalescent sample but in the acute-phase sample as well. The conclusion derived from these tests is that all three individuals had a double infection, the recent one being by yellow fever, the older one by either dengue or Ilhéus in Case 3 and by Ilhéus in Cases 4 and 5.

The examples described seem to indicate that under conditions of natural exposure to Arbor viruses, there exists in man a synergistic or

TABLE 8

GROUP B: NEUTRALIZATION OF YELLOW FEVER VIRUS BY SERA FROM PERSONS WITH ANTIBODIES AGAINST DENGUE VIRUS

Number of sera	Antibodies against dengue, type 2		Antibodies against yellow fever, standard NT	NT antibodies against yellow fever IPER test, Asibi Virus															
				Number of mice inoculated and survivors															
				8									7						
	HI	NT		0	1	2	3	4	5	6	7	8	0	1	2	3	4	5	6	7
15	0	0	0	8		2							3	2						
16	16	16	0				1	1			3	2		1	2	1		2		3

group response similar to the one found experimentally in inoculated guinea pigs.

5. *Possible protective action of group antibodies in human infection.* Exposure to an Arbor virus results experimentally in a certain degree of protection to a challenge by another agent from the same group. Whether a similar situation prevails in man under natural conditions cannot be answered at present. Certain investigations, however, seem pertinent to the subject.*

In a study on the distribution of Group B antibodies among residents of Port-of-Spain, Trinidad, B.W.I., it was found that a high proportion of the population had antibodies against dengue, type 2, and that the proportion of positive reactions increased with age. From the age group 5 to 10 years, 31 sera were selected; 15 of these (TABLE 8) had neither NT nor HI antibodies against dengue 2 (strain Tr 1751), while 16 had both NT and HI antibodies. All these sera were tested by NT test against yellow fever virus, using the standard intracerebral inoculation test in which undiluted sera mixed with 50 to 100 $L.D._{50}$ of virus were injected into mice 3 to 4 weeks old; all 31 sera failed to protect under these conditions. The sera were next tested in an intraperitoneal NT test by inoculation of a mixture of undiluted serum and 50 to 100 $L.D._{50}$ of yellow fever virus, Asibi strain, into mice 3 to 5 days old. The groups of mice inoculated consisted of either 7 or 8 animals (TABLE 8).

Following inoculation of virus mixed with each of the 15 sera that had not shown NT or HI antibodies against dengue, 11 sera failed completely to protect the 7 or 8 mice injected with each mixture; of the other 4 sera, 2 protected 2 of 8 mice inoculated in each case, and 2 protected only 1 of 7 inoculated in each case. All these 15 sera would be considered as negative in the interpretation of the NT test.

The 16 sera that had shown NT and HI antibodies against dengue virus, although still negative in the standard intracerebral NT test against yellow fever virus, behaved differently in the intraperitoneal NT test

*The following studies are quoted with the permission of M. Theiler.

with yellow fever: 5 protected all of either 7 or 8 mice inoculated, and 3 more protected 7 of 8 mice. These 8 sera would be considered as positive in the usual interpretation of a NT test. Of the remaining 8 sera, 3 (2 with 5 survivors of 7 inoculated and 1 with 4 survivors of 8 infected) can be considered as showing partial protection. In no instance did a serum of this group of 16 fail to protect some of the inoculated mice.

There seems to be no doubt, then, that the presence of NT antibodies against dengue virus detected by the intracerebral test (that is, possible prior infection by dengue), and the absence of such antibodies against yellow fever virus (that is, no prior exposure to this virus), resulted in a definite degree of protection of mice against yellow fever virus inoculated peripherally. Whether this NT antibody, demonstrable only by intraperitoneal test, could protect the human donor totally or partially against yellow fever is a matter for further study.

Summary

Arthropod-borne (Arbor) animal viruses can be assembled in sharply defined groups on the basis of serological interreactions detected specifically by hemagglutination inhibition tests. At present, the three such groups that have been characterized have been designated A, B, and C; the indications suggest that additional serological groupings exist. Although several Arbor viruses have not been placed in any of the described serological groups and may remain as isolated entities, the indications are that group similarities are fairly common among many of the Arbor viruses and that the underlying mechanism is probably the result of common antigenic constituents among the members.

As the work with these agents progressed, it became more challenging to determine whether the grouping property of Arbor viruses represented more than a simple academic taxonomy. From the outset there was no doubt that this grouping constituted a helpful device for facilitating identification of new isolates. As the evidence accumulated, however, the different groupings revealed profound relationships to the course of events in natural infections of man and lower animals. Thus, mutual interactions between two viruses of a group can be detected in experimental animals; these result in serological responses beyond the ability of each one separately. The evidence gathered seems to indicate that this is also the case in man naturally infected. In experimental animals, the capacity of an agent to protect against another virus of the same group could be demonstrated. The question whether a human population that has been exposed to infection by a virus will show an increased resistance against invasion by another microorganism of the same group must be left unanswered. In this connection, however, one must note the protection secured by appropriate tests against yellow fever virus with

sera from persons who had presumably been infected previously with dengue. The corollary problem, whether one virus of a group is capable of preventing or modifying infection, not only against itself, but also against all or many other members of that group, (as, for instance, in vaccination), awaits further experimentation. The aforementioned results and observations provide a logical basis for such a notion.

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II. RESPONSES OF PLANTS TO CONCURRENT INFECTION BY TWO OR MORE VIRUSES*

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In nature, concurrent infection of plants by two or more viruses or virus strains is quite common. Probably no natural infection is caused by a single homogeneous strain, but rather by a group of strains differing in pathogenicity. With almost all viruses new variants frequently arise, and a few of these become established in the plant in which they are formed. These variants may persist there and constitute a small part of inocula taken from that plant. Almost without exception, all serious attempts to separate different strains from a naturally infected plant have been successful. One or a few predominant strains probably constitute the bulk of the actual virus material present, but many other minor strains are almost certainly present in smaller amounts. If one could plot the amount of each virus strain present against its relative pathogenicity, the resultant graph probably would resemble a normal distribution curve. The dominant strain or strains are the ones primarily responsible for the disease caused, although symptoms may be modified somewhat by some of the minor strains. The dominant strains constitute the bulk of any inoculum taken from such plants; hence the disease remains constant in serial inoculations. Occasionally, however, one of the minor strains will become established in a newly inoculated plant and a new disease will result.

The relative amounts of the various strains produced in a given plant will depend not only upon the relative amounts of each in the inoculum but also upon how well each is able to compete with the others. Certain strains will predominate because they multiply most rapidly, are the most invasive, or otherwise are best suited for survival. If transferred to another host species, some other strain may be the one best equipped for survival and, sooner or later, it will become dominant.

Infection by two or more distinct viruses that are not related at the strain level frequently occurs. The different viruses may not interfere with each other; each may invade the plant independently of the others and produce its own peculiar effects. This is not always the case, for interaction between such viruses in mixed infections is well authenticated. In a few cases, these interactions resemble those found among related strains, but in most instances they are quite different.

*This paper, illustrated with lantern slides, was the second of three papers presented at a joint meeting of the Section of Biology and the Division of Mycology, December 10, 1956.

Responses of Plants Infected by One Virus to Inoculation with Another Virus

Prior infection of a plant by a virus usually prevents subsequent infection of that plant by other strains of the same virus.[1] This cross protection may be complete or only partial, depending in some degree upon how completely the first strain invades the plant and upon how closely related the second or challenge strain is to the established one. Generally this protection or antagonism is reciprocal, although the degree of protection may be much greater in one direction than in the other. Explanations for this mutual interference generally are based on assumptions that infection by one strain excludes other strains because the first has appropriated or utilized the available multiplication sites, mechanisms, and/or raw materials essential for the multiplication of the challenge strains.

This protective effect seldom occurs among viruses that are not related strains. In almost all cases, a plant infected by one virus retains its susceptibility to infection by a second; however, the level of susceptibility to the second virus may be lessened somewhat by the first infection. McKinney[2] noted that plants infected by any one of certain viruses produced fewer local lesions when inoculated with yet another distinct virus than did comparable healthy plants. This kind of nonspecific antagonism probably results from an effect of the first virus on the general vigor or physiology of the plant. Diseased and healthy plants may be expected to react differently to inoculation with other viruses.

An unusual case of induction of apparent immunity from one virus by prior infection by a distinctly different virus is that of potato virus Y and tobacco severe etch virus. Plants infected by etch virus are protected from infection by virus Y. Bawden and Kassanis,[3] who discovered this interesting relationship, interpret their data as indicating that the etch virus does not interfere with the establishment of virus Y, but rather interferes with its subsequent multiplication.

A second unusual case is that of the cucumber mosaic virus and the tomato aspermy virus. By most criteria, these are not related closely enough to be regarded as strains of the same virus.[4,5] It has been discovered, however, that plants infected by either one are protected from infection by the other.[6] Perhaps the interaction shown by these two exceptional pairs implies either kinship at a taxonomic level not much above that of strain relationship or interaction of an entirely different type than that which occurs between strains.

Responses Immediately Following Simultaneous Inoculation with More than One Virus

When a plant is inoculated simultaneously with two or more viruses to which it is susceptible, all usually will cause infections initially regard-

less of how closely they are related. In some cases, however, one may affect the number of local infections caused by the other. This may be because one virus kills the cells invaded, thereby preventing multiplication and spread of other viruses introduced into the same cells. In other cases, a non-lesion-forming strain may be the one first established in certain sites and may effect local cross protection, thereby excluding a lesion-forming strain of the same virus from such areas.[7]

If a plant is inoculated with a mixed inoculum containing one virus to which it is susceptible and another to which it is not susceptible, the presence of the latter virus seldom has an effect on infection by the former. There are at least two apparent exceptions, one concerned with related strains and the other with viruses not so related. Bean leaves appear to be immune from the ribgrass strain of tobacco mosaic virus, whereas the ordinary strain produces numerous local lesions. Helen Purdy Beale[8] found that when bean leaves were inoculated with a mixture of the two strains fewer lesions formed than when the common strain was used alone. A similar interference has been noted between two distinct viruses.[9] Potato seedling 41956 apparently is immune from potato virus X but reacts to inoculation with potato virus Y by formation of local lesions. Inoculation of the plant with a mixture of the two viruses resulted in strikingly fewer lesions than did inoculation with virus Y alone. When the amount of virus Y in the inoculum was held constant, the number of lesions formed was approximately in inverse proportion to the amount of virus X in the inoculum. The one virus does not inactivate the other *in vitro*; hence, it appears that there must be some kind of reaction between the test plant and the virus to which it is supposedly immune. Perhaps these two examples are cases of extreme hypersensitivity; for example, virus X may infect some cells of the supposedly immune host, but the reaction is so violent that the cells invaded are killed quickly and the virus is thereby localized. If virus Y had entered the same site, it could not multiply sufficiently to move to other cells.

Virus Multiplication and Symptom Development in Plants Infected by One or More Viruses

Once two or more viruses become established in a plant, the subsequent events may follow any one of several patterns. If the viruses are related strains, all may become established and multiply; cells completely invaded first by a particular strain presumably will produce only that strain. It is not known whether two or more related strains can become established and multiply in the same cell, but recent work by Benda[10] has shown that two strains can be introduced through the same cell. In plants infected by several strains, the various strains multiply in direct competition with each other. Symptoms are intermediate in severity; there exists no concrete evidence to show that they are ever

more severe than those induced by the most pathogenic strain alone or that they are ever less severe than those induced by only the least pathogenic strain.[5] This situation – and the fact that symptoms induced by a given virus in a given host generally are related directly to the concentration of that virus – strongly suggests that the total virus content of the plant is intermediate between the extremes reached by the component viruses in single infections. This content probably does not exceed the limits reached by each strain separately. This viewpoint is consistent with the available data.[1]

One might assume that there is somewhat the same kind of competition for synthesizing mechanisms or for plant metabolites among distinctly different viruses. No doubt nonspecific competition does occur to some extent in many cases, but in general it is not sufficiently important to cause striking suppression of multiplication of the component viruses or of the symptoms thereby induced. In some cases, there is a more specific type of interaction, varying from a complete suppression of one virus by the other to a marked synergistic pathogenic affect.

Bawden and Kassanis[11] discovered that tobacco severe etch virus prevented the multiplication of potato virus Y or of a henbane virus in simultaneously inoculated plants and replaced them in plants in which either was established. Mild etch virus, a strain closely related to severe etch virus, only partially suppressed the other viruses. In both cases symptoms were modified accordingly. This displacement or suppression was explained on the basis that etch virus so affects the metabolism of the cells that they no longer produce some material or enzyme system that is normally present and essential for the multiplication of the displaced viruses.

At the other extreme, there are many cases in which symptoms induced by two viruses together are much more severe or of a different type than would be anticipated on the basis of what each virus does alone. Where such cases have been studied, it has been found that the doubly infected plants contain a strikingly greater amount of one component virus than that contained in comparable singly infected plants.

The first quantitative work on this type of infection was done by Bennett.[12] When tomato plants were inoculated with dodder latent mosaic virus, the resultant disease passed through a necrotic phase that was followed by recovery and a marked decrease in virus concentration. When these recovered leaves were inoculated with tobacco etch virus or tobacco mosaic virus, symptoms characteristically induced by the original virus again appeared; the concentration of dodder latent mosaic virus increased markedly and remained at a level much higher than that in comparable plants infected by this virus alone.

Rochow and Ross[13] have studied several double infections involving potato virus X, a virus that appears to be unusually sensitive to the presence of other viruses. We worked with a strain that produces no

obvious symptoms in tobacco, the host species used. Simultaneous inoculation with this virus and potato virus Y resulted in symptoms much more severe than those caused by either virus in single infections; this was associated with a virus X concentration as much as ten times greater than that in comparable singly infected plants. When tobacco etch virus was the other member of the pair, symptoms again were more than additive; the virus X concentration was as much as six times as great as that in the controls. Similarly, tobacco mosaic virus plus virus X caused marked synergistic effects, and the concentration of the latter was at least four times that of the controls. The synergistic effect was less marked than the foregoing when cucumber mosaic virus and virus X were paired; this was associated with a twofold increase in virus X. In contrast, when virus X was paired with either alfalfa mosaic virus or tobacco ringspot virus, symptoms were no more than additive, and the virus X concentration was either the same as (when with alfalfa mosaic virus) or less than (when with tobacco ringspot virus) that in singly infected plants. Of these pairs, assays for the other members of the pairs were made only in the case of virus Y and tobacco mosaic virus. The concentration of virus Y was the same in doubly and singly infected plants. That of tobacco mosaic virus was slightly lower in doubly infected plants than in those singly infected. The results with tobacco mosaic virus and virus X are in accord with those reported by Ishii.[14]

The foregoing data on alfalfa mosaic virus and virus X suggest that virus pairs that induce only additive symptoms in double infections multiply entirely independently of each other. This is probably the case with many such double infections, but it seems that in others one or both viruses may exert minor effects on the multiplication of the other. In recent work with infections that reportedly resulted in additive symptoms, Garcés-Orejuela and Pound[15] found that infection by cucumber mosaic virus suppressed multiplication of tobacco mosaic virus during the initial stages of infection but not subsequently; multiplication of the former virus appeared to be stimulated early in the infection process. Later, however, the doubly infected plants contained slightly lesser amounts of this virus than did comparable singly infected ones. Slightly more tobacco mosaic virus was found in plants also infected by tobacco ringspot virus than in plants infected only by tobacco mosaic virus. This stimulated multiplication of tobacco mosaic virus in the presence of tobacco ringspot virus is of particular interest, since Kunkel[16] has reported that tobacco plants infected by tobacco ringspot virus are spared from the severe effects ordinarily produced by tobacco mosaic virus.

The striking interaction of viruses X and Y led us to undertake further experiments with this pair. As already indicated, we found that when tobacco plants were inoculated simultaneously with the two viruses, the concentration of virus X reached a level far beyond that reached in

singly infected plants. It became evident, however, that the level reached in doubly infected plants varied with the stage of the disease.[17] In inoculated leaves, the ratio of the virus X concentration in doubly infected plants to that in those singly infected was about two. During the acute stage of the disease, in leaves that were expanding rapidly when invaded, this ratio was as high as ten. This stage also is the stage of maximum symptom development. Thus, a tenfold increase in virus X concentration was associated with a marked increase in the severity of symptoms. During the chronic stage of the disease, when symptoms were somewhat less severe than during the acute stage, there was up to four times as much virus in doubly infected plants as in singly infected ones.

The foregoing results were based on assays by local lesions in *Gomphrena globosa* L., the standard test plant for this virus. Such assays usually give information on relative amounts of active virus, but they do not necessarily measure total virus material and are subject to error because of the possible presence of materials that affect infectivity of the virus or the sensitivity of the test plant. Accordingly, we made parallel assays by an independent method – by particle counts with the electron microscope.[18] The results achieved by the two procedures agreed very closely, for both methods indicated that leaves sampled during the acute stage of the disease contained ten times as much virus material when doubly as when singly infected.

When plants systemically infected by virus Y were inoculated with virus X, the concentration of virus was not nearly as high, nor the symptoms as severe, during the acute stage of the disease as when inoculations with the two viruses were simultaneous. In contrast, when inoculation with virus X preceded that with virus Y, the result was much the same as with plants inoculated simultaneously with the two viruses; very severe symptoms developed in certain leaves, and these leaves contained ten times as much virus as did comparable singly infected ones.

It thus appears that most of the excess virus X is formed during the rapid phase of Y multiplication. In other words, the amount of excess virus X formed during any period seems to be roughly proportional to the amount of virus Y synthesized during that period. Experiments on the distribution of these viruses between veinal and interveinal tobacco leaf tissue provided further evidence that the excess synthesis of virus X is a direct result of virus Y synthesis. More virus Y was found in veinal than in interveinal tissue, and the relative change in virus X concentration resulting from double infection was greater in veinal than in interveinal tissue.

These data make it evident that during the synthesis of virus Y there is brought about some change that results in a temporary increase in the amount of virus X synthesis that plant tissue will support. This change could be caused by some material that is formed as a by-product of virus

Y synthesis or that accumulates as the result of a disruption of normal metabolic cycles brought about by infection by virus Y. In this connection, it is becoming increasingly evident that infection by viruses may result in the formation of numerous anomalous products or in the accumulation of unusually large amounts of materials normal to the plant. If such a product is formed, it could act in at least two ways.

First, it may be the virus X "building block" in shortest supply, that is, the one that normally limits the amount of virus X formed. If this is the case, then infection by virus X must result in at least a tenfold increase in the availability of this material.

Second, the substance may act to nullify the action of some other substance that normally limits synthesis of virus X or to unblock some enzyme system that becomes obstructed during X synthesis. This causes one to speculate on the mechanism responsible for the eventual deceleration in the rate of virus synthesis in the absence of obvious symptoms. It generally is assumed that virus synthesis is slowed because of complete or nearly complete depletion of the materials from which the viruses are synthesized. It is difficult to see how this could occur without exerting some obvious effect on the host plant, for virus synthesis and normal protein synthesis appear to draw on the same pool of raw material. Perhaps a by-product of virus X synthesis acts to inhibit subsequent multiplication, much as "staling products" limit the growth of many microorganisms. If this is true, it may be that a product formed during synthesis of virus Y serves to nullify the action of these "staling products."

A slightly different view holds that these "staling products" are used directly during the synthesis of virus Y. Still another contention is that infection by virus Y merely increases susceptibility to virus X by making available more sites at which virus X can become established and multiply.

At present we know of no data that point directly to any particular hypothesis. The problem seems subject to experimental attack, however, and perhaps a study of multiple infection will provide another approach to the solution of the question of the mechanism of the multiplication of viruses.

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III. THE RELATION OF ARTHROPOD-BORNE VIRUSES TO THEIR INVERTEBRATE HOSTS*

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Although we have known of the arthropods' role in the transmission of viruses for more than half a century, we still do not understand the fate of the virus in the vectors. For many years biologists have accepted the view that some of the viral encephalitides pathogenic for vertebrates multiply in their insect vectors as well as in their vertebrate hosts. It was difficult to prove this multiplication in the presence of a large amount of ingested virus, but when the amounts ingested were kept small, multiplication was relatively easy to detect. It was appreciated, of course, that the virus was suddenly subjected to two very different environments, but this presented no conceptual difficulty because even malarial sporozoites and filarial worms were known to withstand sudden injection from the mosquito's salivary gland into the vertebrate bloodstream with a consequent marked and sudden change in environmental conditions.

On the other hand, there was, until quite recently, considerable resistance to the idea that viruses that infect plants could also undergo multiplication in insects. The proof of this is, I believe, one of the most remarkable discoveries in biology in recent years, and the consequences that flow from it are far reaching (see Maramorosch, 1955). Viral synthesis undoubtedly requires a complex set of conditions, and it is now clear that these can be provided by hosts as notably different as a leafhopper and a potato plant. In this respect, therefore, these viruses must be considered highly versatile.

There is one aspect of this versatility that has not received much attention. This concerns the fact that the alternation between hosts must exercise a stringent selection within the virus population. Undoubtedly this aspect is a factor contributing to the observation that those viruses that multiply both in their arthropod and in the mammalian or plant hosts generally occur in fewer strains than the viruses transmitted without the intervention of a vector. It is known that when certain arthropod-transmitted viruses are transmitted by means other than by their normal vectors (as, for example, by grafting), they may change. On several occasions, Black (1953a) has shown that the resulting virus cannot be transmitted by its original vectors. It may have lost the ability to multiply in the arthropod vector; there are other possible explanations which currently are being investigated (Black, Whitcomb, Wolcyrz, unpublished).

*This paper was the third of three papers presented at a joint meeting of the Section of Biology and the Division of Mycology, December 10, 1956.

There is a paradox associated with this versatility. In some respects viruses may be highly specific. It has been known for many years that the virus of aster yellows is transmitted by the leafhopper *Macrosteles fascifrons*. The virus multiplies in this leafhopper, but it will not multiply in, nor can it be transmitted by, *Macrosteles laevis* from Europe. However, *laevis* is so closely related to *fascifrons* that even well-trained taxonomists may be unable to distinguish the two (Maramorosch, 1956b). The reasons for this specificity or lack of versatility are quite unknown, and an explanation is not made easier by the fact that the related Californian strain of aster yellows virus has been shown by the Berkeley group to be transmitted by about twenty-five species of leafhoppers belonging to at least eleven genera (see Day & Bennetts, 1954). Likewise, although the plant-host range of this virus, and of others resembling it, is frequently quite wide, there are many plant species in which the virus is quite unable to multiply.

There is, then, remarkable vector specificity in some viruses. This is shown particularly well by the work of Black (1944) on the two strains of potato yellow dwarf virus; the New York strain is transmitted by four species of *Aceratagallia* and rarely by *Agallia quadripunctata* and *Agalliopsis novella*. The New Jersey variety is transmitted by two species of *Agallia* and rarely by *Agalliopsis novella*, but not by a species of *Aceratagallia* that transmits the New York variety. One finds a similar situation in the animal viruses; for example, dengue fever is transmitted by several species of the genus *Aedes*, but not by *Culex* or other related mosquitoes. On the other hand, it has now been shown that yellow fever is transmitted in Africa and in South America by a very large number of species of mosquitoes belonging to several subfamilies. Of course, one can never tell how much of this specificity of such viruses as dengue would be reduced were more work to be done on them but, from present knowledge, dengue appears to be a relatively specific virus for both its vertebrate and invertebrate hosts.

It should also be noted that records in the literature of the inability of an arthropod to transmit a virus must often be accepted with caution. Strains of arthropods differ in their efficiency as vectors and require an appropriate indicator host to demonstrate the presence of virus infection. Transmission to vertebrates may occur without frank symptoms, but produce only a serological response, and this has not always been sought. Records of vector ability are, therefore, often more reliable than records of the failure of an arthropod to transmit a particular pathogen, although there are also in the literature many records of vector ability that seem unfortunately to be erroneous.

The explanation of vector specificity appears to be that, in the arthropod, there are a series of barriers, one to virus penetration of the gut, another to virus multiplication in the cells, and perhaps also another to

the penetration of the virus into the salivary glands, whence it can be reinjected into the next host. The nature of the barriers, however, is not understood, and their very existence has yet to be proved, although there is evidence for the existence of each.

Genetic studies of differences in vector efficiency have demonstrated marked variations in this quality in leafhoppers transmitting some leafhopper-borne viruses (Black, 1953b), and there are indications that such differences exist in mosquitoes transmitting Rift Valley fever (Gillett and Mims, 1955). It seems probable that such differences between strains are more important than is generally believed, and that investigators would do well to take these differences into consideration when undertaking work on arthropod transmission. One of the unanswered questions is whether these differences in vector specificity would be removed if the tissues were in culture but, so far, no insect tissues have been maintained in continuing culture, and comparatively little work has been done (except for that of Trager, 1938, and Maramorosch, 1956a) on the growth of viruses in insect tissue culture.

The hypothesis, originating with Theobald Smith, that long association between virus and host results in a reduction in pathogenicity clearly seems to be only partly true. Although there is obvious survival value for a virus to become symbiotic rather than so efficiently pathogenic that it kills all its hosts, it seems certain that the pathogenicity of a virus to a new host is purely a matter of chance. In what way, except on the basis of chance, could one explain the fact that the strain of Brazilian myxoma virus that invaded France, and the strain that came from South America via England and swept over the southeastern part of Australia, were so highly pathogenic for the European rabbit? We now know that a strain of somewhat reduced virulence has replaced the highly infectious strain in much of Australia. We believe this to be due to the fact that, although the concentration of virus in the skin is maintained, the period during which viruliferous lesions are available for mosquito vectors to acquire virus is somewhat increased (Fenner, Day, and Woodroofe, 1956). This very plausible hypothesis did not explain the fact that in Britain attenuated strains seemingly have appeared less frequently than they have in Australia. However, in Britain it is thought that the rabbit flea *Spilopsilus* (which never reached Australia, although it is now to be introduced) is the vector, and it is known that this vector leaves the rabbit generally only after the death of the host. Thus, as F.N. Ratcliffe (who is in charge of the Wildlife Survey Section, Commonwealth Scientific and Industrial Research Organization, Australia) has pointed out in a personal communication, an increase in survival time of a few days would not permit more vectors to feed on that host and would thus invest the virus with no selective advantage over the virulent parent strain.

The fact that a difference so apparently trivial can have such a marked effect on the epidemiology of the disease and on the virulence of strains found in the field is a matter of considerable biological interest. It should be stressed that often there is no correlation between the virus content of a host and symptom expression. Often completely symptomless reservoirs are effective virus sources, and the possibility of seed transmission and of transovarial or transplacental transmission in such hosts makes the task of the epidemiologist one of great complexity.

As I have mentioned, there are two strains of aster yellows virus, the Californian strain and the Eastern strain, that can readily be distinguished by the production of distinct symptoms in different plants. The Californian strain and the Eastern strain protect against each other in plants, and Kunkel (1955) has shown that they also protect against each other in the vector. When the leafhopper is infected with one strain, it is immune from infection by the other. The reverse situation has been known for some years to exist in sugarbeet curly top (Giddings, 1950). A beet leafhopper that is carrying one strain of curly top virus can acquire and transmit another strain. The same finding has been made recently by Chamberlain (1956) with Eastern equine encephalitis and Western equine encephalitis virus diseases. In one experiment the Eastern strain and, one week later, the Western strain, were fed to a mosquito; the mosquitoes were then incubated for two weeks. Each virus could be tested for in the presence of hyperimmune serum of the other. Both were transmitted together by a single vector in 94 per cent of the cases, and there was evidence that there was as much of each virus present as if the mosquito had been infected with one strain alone. In this instance, therefore, there was no interference between the two viruses. Of course, it may be that these two viruses are less closely related than has been thought and that they do not, in fact, compete within the host for either penetration sites or metabolites. Maramorosch (1957) has described an intermediate condition in which two strains of corn stunt virus can either interfere in the vector or not, depending upon the interval that elapses between feeding on the two strains.

It was reported by Varma (1955) that a single whitefly was able to transmit two distinct viruses, but it is reasonably certain that these viruses are unrelated.

Observations on the presence of virus in the vectors raise questions of the role of humoral immunity in insects. A good deal of work has been done in the attempt to demonstrate whether arthropods do or do not produce antibodies, using methods that have been known to be sensitive for demonstrating them in vertebrates. The results have been almost uniformly unrewarding. This is hardly surprising, as there is no reason to suspect that an immune response of arthropods would result in the production of

substances detectable by the methods developed for mammalian immunology. On the other hand, some recent results of Briggs (1956) indicate that an acquired tolerance to bacteria did develop within twenty-four hours after inoculation into a caterpillar and continued through the duration of larval life. This antibacterial substance was extremely stable to heat. It was never demonstrated after feeding the inoculum. Briggs has also shown that nonspecific antibacterial substances are present in low concentration in normal insects and that these may originate from blood cells. The indications are that the protective mechanisms of insects are not likely to be understood by the methods found suitable for study of vertebrate systems, and further knowledge must await the development of methods particularly adapted to the insect mechanisms. In view of this lack of basic information, it is not unexpected for us to find some contradictory or apparently contradictory data on the occurrence of interference between two plant viruses in a leafhopper vector and the complete absence of such interference between the two encephalitides previously mentioned. Until we know something about the mechanism of attachment of these viruses to the host cells, and the tissues and substrates utilized, any conclusions would be premature. Nevertheless, the existence of these two situations indicates that results of great interest will emerge from further study of multiple infections, and it may be predicted that such results would have an interest far beyond the special field to which they directly apply.

It has generally been believed that arthropod vectors of viruses show no pathogenic symptoms resulting from the viruses that they ingest and transmit. It is, therefore, of great interest to note that Littau and Maramorosch (1956) have shown recently that aster yellows virus is able to cause distinct cytopathogenic effects in the nuclei of fat body of a very high percentage of infected leafhoppers. Preliminary results with other viruses and vectors have not yet uncovered another comparable instance.

One of the most interesting observations in the study of arthropod vectors of viruses concerns transovarial infection, in which the virus passes through the egg of the female parent, and the offspring in varying percentages are infected with the virus and capable of transmitting it. This infection has been known in several leafhopper-borne viruses for some time, but it has not been found in any of the aphid-borne viruses or, in fact, in any of the plant pathogens other than in the leafhopper-borne diseases. Among animal diseases transovarial infection occurs in a number of *Rickettsiæ*, and many of the symbiont-like organisms found in insects are transmitted through the egg. There are records of transovarial infection in ticks and mites of certain encephalitis-like diseases, but the earlier results have not been repeatable in the hands of later workers.

The mechanism by which some and not all of the offspring are infected in instances of transovarial infection is not understood. It may be that

the virus multiplies in limited amount or it may be that it is able to penetrate into eggs only in certain stages of development. In Rocky Mountain spotted fever, transovarial infection is not in itself a sufficient means of maintaining the pathogen through the unfavorable seasons; obviously, therefore, the tick is not an adequate reservoir of infection (Price 1954a). So far as is known, there is no example of transovarial infection in mosquitoes, but this possibility should not be entirely ruled out, as there is one example in which eggs of infective *Mansonia* mosquitoes were found to harbor virus (Kissling *et al.*, 1955). Similarly, if larvae of *Aedes aegypti* are fed large amounts of yellow fever virus they may be able to carry it through to the adult stage, and it has been demonstrated that the adult is able to transmit the disease. There seems to be no barrier, therefore, to transovarial infection and the possibility needs additional careful study.

So far no one has discovered transovarial infection in the mite-borne phytopathogens. This is a relatively neglected group of virus diseases and, as yet, very little work has been done on the means by which the mite is able to transmit the disease. By analogy with the situation in the vertebrate pathogens, however, it is possible that transovarial infection could occur in these arthropods, and a study of this would be of great practical and theoretical interest. Price (1954b) has recently shown that, in ticks transmitting Rocky Mountain spotted fever *Rickettsiae*, the molting of the tick has an effect on the virulence of the pathogen that the tick excretes. This is the first observation of an effect of the arthropod on the pathogen that it transmits. Complete loss of virulence suggests the possible occurrence of latency in the vector. In certain leafhopper-borne viruses the offspring of infective females may fail to transmit the virus, but their progeny may be vectors without access to a new source of virus (Grylls, 1954). The form of the virus in the intervening generation is not known. Until methods are available for assaying "provirus" and for distinguishing "masking" from low concentrations (Beard, 1956), such studies will be impossible. In the polyhedral viruses latency involves the transmission for a number of generations of the virus in a form that is not detectable in the offspring by ordinary techniques, but virulent virus appears when the larvae are kept under unfavorable conditions, when it may then wipe out the entire population. It may be that the virus is being transmitted as virus nucleic acid without its protein protective covering. The phenomenon of latency should, I think, be distinguished from the mere absence of symptom expression. So-called "latent" viruses, for example, are frequently described among plant pathogens but, in my view, this is not a proper use of the term "latency," as these plants are often merely symptomless carriers. Some so-called "resistant" varieties of plants are sometimes excellent sources of viruses, their so-called "re-

sistance" being due only to their absence of symptom expression. I suggest that viruses present in the absence of symptom expression be referred to as "masked viruses," and that the term "latent virus" be confined to those instances where the virus seems to be present in a state differing from the usual form – perhaps equating it to the lysogenic form of bacteriophage. The difficulty is, of course, to differentiate in practice between the two, and it may be that true latent virus in this sense does not exist elsewhere than in phage.

The suggestion has been made (Maramorosch, 1955) that some arthropod-borne viruses may actually have originated in the vectors. If we make the reasonable assumption that the virus was not immediately infectious for its plant or vertebrate hosts, this suggestion presupposes either that the viruses were transovarially transmitted in the vectors, or that they had a vector between the arthropods. Various spiders, reduviids, and other insects have been considered as possible alternative hosts for yellow fever. The work has never, however, given any indication that the species tested were vectors (Gillett, 1955). Transovarial infection appears to be more frequent in arachnids than in insects, and this has led to the suggestion that they may be involved as vectors or reservoirs of many viruses.

The role of ticks and mites in transmitting viruses is a field in which new and interesting information is accumulating rapidly, but no one has yet studied the possible role of the many arthropod-inhabiting mites in transmitting pathogens from arthropod to arthropod, although the suggestion that they should be considered has been proposed (Hurlbut, 1956). The hypothesis is obviously well worth careful study.

It has been possible in this rapid survey only to indicate broad outlines of arthropod-vector relationships. I have attempted to show that in some respects these viruses are both "versatile" and "conservative." I have stressed the fact that we have no idea of the reasons for this, but that study of these phenomena is bound to provide keys to many fundamental properties of viruses. Such a study, I believe, will have important lessons for biology in general.

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SECTION OF MATHEMATICS AND ENGINEERING

ANALYSIS OF SOME COOLED-STRUCTURE PROBLEMS BY ELECTRICAL ANALOGY*

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Heat-flow and energy-transport problems encountered in engineering are often of such a complex nature that orthodox analytical methods of a mathematical, graphical, and numerical nature prove inadequate. As with all particular aspects of applied mechanics analysis, the solution of thermal-energy flow processes is fraught with many complications; under these circumstances recourse to analysis via the resistance concept of energy flow offers distinct advantages. In terms of this concept, processes may be simulated by electrical flow with its attendant circuitry, that is, by electrical analogy. Such analysis by electrical analogy is necessarily predicated on the acceptance of the basically assumed data; accordingly, as a computer technique, the procedure develops results that may be interpreted only in terms of the basic facts, as in the case of any other modern-day computer.

In the application of the electrical analogy procedure, under steady-state or equivalent conditions, use is made of the geometrical sheet-analogue methods developed by the author in conjunction with variegated conduction problems,[1,2,3,4] as well as of resistance network methods.[5,6,7] In the geometrical approach, by use of uniformly electrically conductive sheet material, properly modified under circumstances to deal with multicomponent-material structures, the electrical model facilitates determination of isopotential patterns on a two-dimensional basis that permits ready translation into thermal-circuit (heat-flow) results. This is the model technique utilized in the first of the three problems treated; it is used similarly in the second and third problems, except that equivalent electrical resistance networks are employed in the simulation of the thermal-energy flow processes treated.

In the translation of electrical model results to thermal values, use is made of the potential difference ratio, c, defined:

$$c = \Delta e_x / \Delta e \qquad (1)$$

where Δe_x = electrical potential difference between datum and point x;
Δe = electrical potential difference, overall, and is representative of the over-all temperature difference of the thermal case.

*This paper, illustrated with lantern slides, was presented at a meeting of the Section on December 21, 1956.

From the electrical value of c, equivalent temperatures may be established to correspond with imposed temperature conditions:

$$t_x = t_o + c\Delta t \qquad (2)$$

where t_x = temperature at a given point, degrees F.;
t_o = temperature at datum or reference point, degrees F.;
Δt = over-all temperature difference between top or energy-source temperature and datum (energy receiver or "sink" temperature).

The three cooled-structure problems dealt with in this presentation are as follows: (1) performance predictions for a space-cooling concrete slab with imbedded cooling pipes; (2) the wet-bulb process, involving combined heat transfer and vapor diffusion; and (3) heat flow analysis of a cooled-structure complex.

Performance Predictions for a Space-Cooling Slab with Imbedded Cooling Pipes

The use of a building structure slab containing imbedded pipes sometimes proposed for the cooling of enclosed spaces involves heat transfer through both floor and ceiling surfaces. For a given slab construction and pipe-air temperature difference, the variation of the slab temperatures and the magnitude of the heat flow depend primarily on pipe size and spacing, material conductivity, and the value of the effective heat-transfer surface conductance. The minimum surface temperatures will control the maximum permissible relative humidity for the space air without surface condensation.

The cooling of building enclosures by means of imbedded cooling pipes presumably has been suggested by present-day discussions on, and wide applications of, radiant panel heating for such spaces. Herein the flow of a cooling medium through the pipes imbedded within a slab structure reduces the temperature of the boundary surfaces below that of the space air and thus creates the operating temperature difference for convection and radiant effect. Under these circumstances, both the ceiling surface and the floor surface may constitute, in varying degrees, the effective cooling areas for the heat transfer.

When cooling pipes are imbedded within a slab structure, they set up a complex condition of heat flow paths the surface temperatures and heat flow of which difficult to predict. Especially important, if we are to predict conditions of condensation under circumstances of humid air, is a knowledge of the variation of the surface temperature over the expanse of slab. In addition, knowledge of the probable surface temperatures is necessary for the prediction of heat transfer performance at the surfaces. The heat transfer that takes place involves both air convection and surface radiation; both of these are concerned directly with the surface temperature.

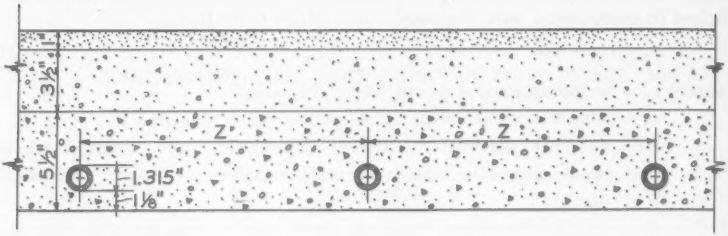

FIGURE 1.

FIGURE 1 represents the structural arrangement investigated for this study. Herein is shown a 10-in. multilayer floor slab in which are imbedded pipes of 1.315-in. diameter at a distance of 1 1/8 in. from the ceiling surface. The construction of the slab is as follows: 5 1/2-in. concrete, 3 1/2-in. cinder fill, and 1-in. cement surface finish at the floor. The specific objective was to determine the influence of pipe spacing and surface conductances on the resultant surface temperatures and on the over-all steady-state heat flow. The pipes are presumed to carry a refrigerating medium, thus lower in temperature than the air of uniform temperature above and below the slab.

The following general relationships may be set up:

$$h_a = h_c + h_r \tag{3}$$

where h_a = total air conductance, in Btu/(hr.-sq. ft.-degrees F.)
h_c = convection air conductance, Btu/(hr.-sq. ft.-degrees F.)
h_r = equivalent radiation conductance – the radiant heat exchange in Btu/(hr.-sq. ft.) divided by a temperature difference, thus Btu/(hr.-sq. ft.-degrees F.), and quite dependent on temperature levels.

Also: $H = \Delta t/R; \quad \Delta t = HR; \quad R = \Delta t/H$ (4)

and $q = HA = A\Delta t/R$ (5)

where H = heat-flow rate per unit of area, Btu/(hr.-sq. ft.)
Δt = over-all temperature difference, degrees F.
R = thermal resistance per unit of area, degrees F./[Btu/(hr.-sq. ft.)], that is, temperature difference degrees F. per unit of heat flow rate H.
q = gross heat flow, Btu/hr.
A = gross heat-flow reference area, sq. ft.

In addition:

$$R_a = 1/h_a \tag{6}$$

$$R_w = L_w/k_w \tag{7}$$

where R_a = equivalent thermal resistance of the air boundary, degrees F/[Btu/(hr.-sq. ft.)]

R_w = resistance of wall or slab material of uniform composition, degrees F./[Btu/(hr.-sq. ft.)]

L_w = thickness of wall or slab material, inches.

k_w = thermal conductivity of wall or slab material, Btu/(hr.-sq. ft.-degrees F/in.)

As previously indicated, for a given value of h_a there is a corresponding value of thermal resistance R_a. Accordingly, for a given conductivity of slab material, there is then some equivalent thickness L_e of slab material that will give the same resistance R_e to heat transfer as the air boundary:

$$R_a = 1/h_a = R_e = L_e/k_w \tag{8}$$

Hence: $L_e = k_w/h_a$, in. (9)

As noted above, due to the cooling action of the imbedded pipes, the actual temperatures existent on the slab surfaces will be lower than that of the space air. Of immediate concern, therefore, with humid space air is the question of condensation on the surfaces due to temperatures

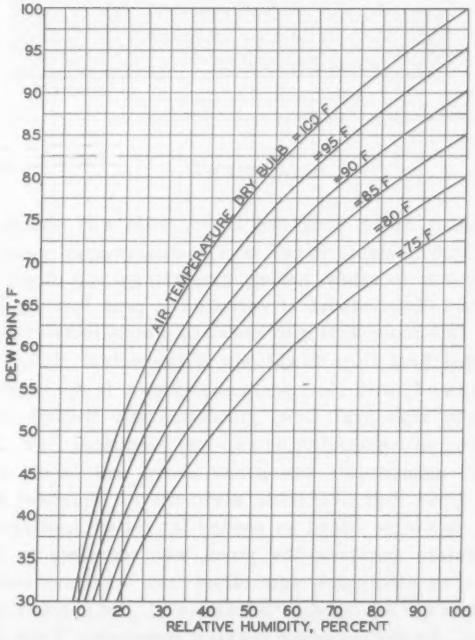

FIGURE 2.

below the dew point of the air. Of further direct concern, also, since the pipes will be still further below the air temperature than the surfaces, is the vapor porosity of the slab material, inasmuch as the water vapor in the air will tend to diffuse through the material and will tend to condense when its lower dew point is achieved. Under these circumstances, unless adequate vapor-barrier protection is provided, the slab will become laden with liquid moisture, its thermal properties may change, and it may suffer severe physical damage. This is an important, although separate, aspect that must be considered. FIGURE 2 shows a typical variation of dew point with relative humidity, for different values of air temperature (dry bulb).

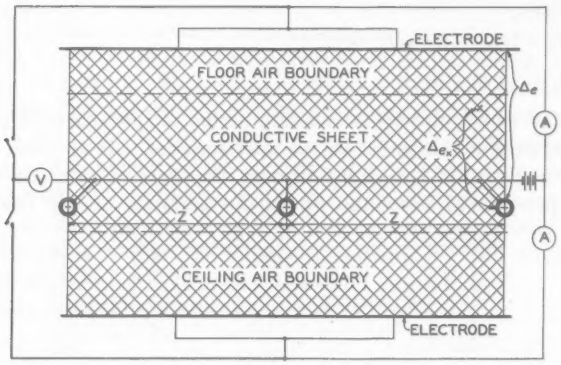

FIGURE 3.

A slab section 32 in. long and 12 in. wide was chosen for this investigation, with analysis based on two-dimensional steady-state flow. The objective was to study, among other things, the effect on heat flow and temperature distributions of variation in pipe spacing Z from 32 in. downward. A full-scale analogue model was constructed to represent the two-dimensional heat flow conditions of the actual slab. This is illustrated in FIGURE 3, with electrical connections. In the electrical analogy model, two-way slab heat flow, simultaneously through the upper and lower surfaces, was simulated by two-way electrical flow. Electrodes to represent the ambient air conditions above and below are shown in FIGURE 3, along with electrode elements to represent the imbedded pipes; extra ones were added as needed for varied spacings. The 5.5-in. layer of concrete encasing the pipes was regarded as the reference material. The air electrodes were offset from the outer slab surfaces in accordance with the equivalent "thickness" L_e determined by EQUATION 9 and using k_w for the reference material (concrete). Thus, the electrical model provides, through adaptation of Ohm's law, an electrical flow

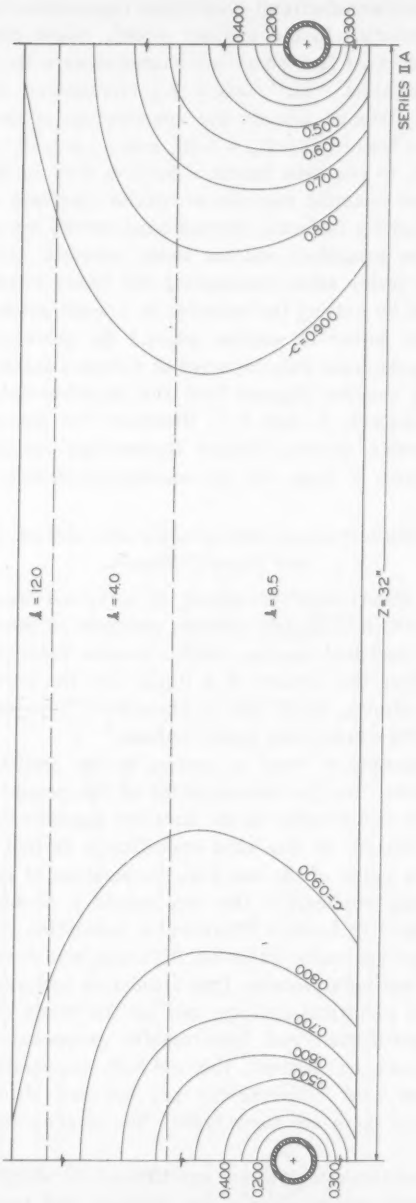
0.400
0.200
0.300
SERIES II A
0.500
0.600
0.700
0.800
C=0.900
λ=12.0
λ=4.0
λ=8.5
Z=32"
C=0.900
0.800
0.700
0.600
0.500
0.400
0.200
0.300

FIGURE 4.

equivalent of the thermal-circuit conditions represented through thermal resistances. Simulating in the analogy model, linear distances on the electrically conductive flat sheet are commensurate to thermal resistances. The equivalent "air" length L_e represented directly by the electrode offset distance permits the construction of the model of the heat-flow path to scale (with $k_w = 4.90$, and $h_a = 1.50$; $L_e = 3.27$ in.).

For the model, to preclude lateral electrical flow in the air-boundary section, the sheet material was slit at regular intervals normal to electrodes. To account for differing thermal conductivity between the cinder fill (relatively an insulator) and the basic concrete, the electrical resistivity of the model area representing the lower conductivity cinder fill was modified by cutting the material in a mesh pattern, a procedure described by the author in another paper.[1] By galvanometer-slidewire means, relative potentials were obtained at different points.

To typify the results, figures from the experimental program,[4] are shown as FIGURES 4, 5, and 6 to illustrate the pipe-spacing effect; FIGURE 7 presents typical surface temperature results, coordinated through EQUATION 2 from the galvanometer-slidewire results for c.

The Wet-Bulb Process, Involving Combined Heat Transfer and Vapor Diffusion

Simultaneous steady-state occurence of both heat transfer and vapor diffusion, involving a vapor-gas mixture, presents an interesting problem for analysis by electrical analogy. Such a system would be one in which vapor diffuses from the surface of a liquid into the surrounding atmosphere of a flow stream, while heat is transferred between the vapor-gas atmosphere and the evaporating liquid surface.[9]

A specific example of such a system is the well-known wet-bulb-thermometer process for the determination of the properties of a vapor-gas mixture, with heat transfer in the direction opposite to vapor transfer. The vapor-gas mixture at this time specifically is that of water-vapor and air, with the value of the wet-bulb temperature of prime importance in air-conditioning studies. At the very outset it should be noted that the principal objective here is to present a simulation technique involving both heat flow and vapor diffusion. Although here developed primarily in terms of the wet-bulb process, this simulation technique is presented as an additional electrical-analogy tool for the study of other complex combined vapor-diffusion and heat-transfer processes. Certainly, independent of analogue studies, the wet-bulb temperature of a water-vapor-air mixture most conveniently may be read off a pyschrometric diagram, of which there are many types. One of them is represented by FIGURE 8.

Under the conditions of dynamic equilibrium, in which a nonsaturated vapor-gas mixture passes over a wet surface, the temperature of the

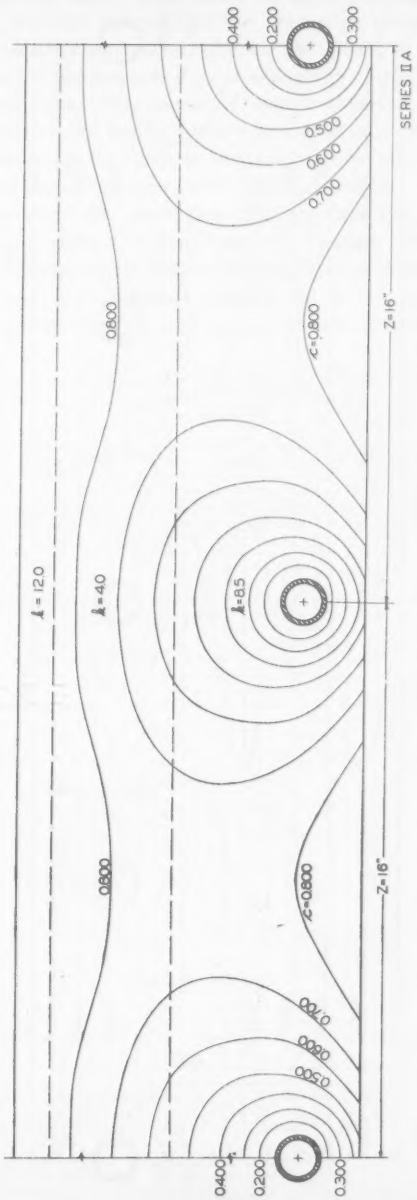
SERIES II A
0.400
0.200
0.300
0.500
0.600
0.700
0.800
Z=0.800
Z=16"
k=120
k=40
k=8.5
0.800
Z=0.800
Z=16"
0.400
0.200
0.300

FIGURE 5.

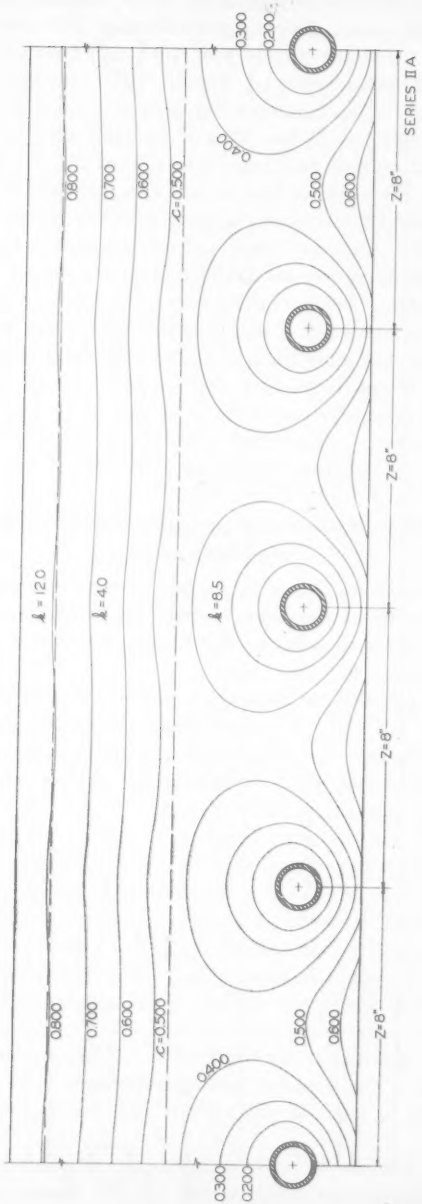
SERIES II A
λ=12.0
λ=4.0
λ=8.5
C=0.500
C=0.500
0.800
0.700
0.600
0.500
0.400
0.300
0.200
Z=8"
Z=8"
Z=8"
Z=8"

FIGURE 6.

latter stabilizes at some temperature between the mixture "dry-bulb" temperature t_d, and the "dew-point" or saturation temperature t_s of the mixture. This equilibrium temperatore for the liquid is generally known as the "wet-bulb" temperature t_w corresponding to the conditions of t_d and t_s. At equilibrium, the amount of heat transferred from the flowing vapor-gas mixture to the liquid surface is essentially proportional to the temperature difference Δt between the gas mixture and the liquid interface. It simultaneously represents the amount of vaporization-heat necessary to produce the vapor that diffuses from the surface into the mixture vapor-gas stream flowing past it. Though various other viewpoints, such as vapor-concentration difference, may be employed, the rate of vapor flow may be considered proportional to the vapor-pressure difference ΔP existing between the vapor pressure corresponding to the

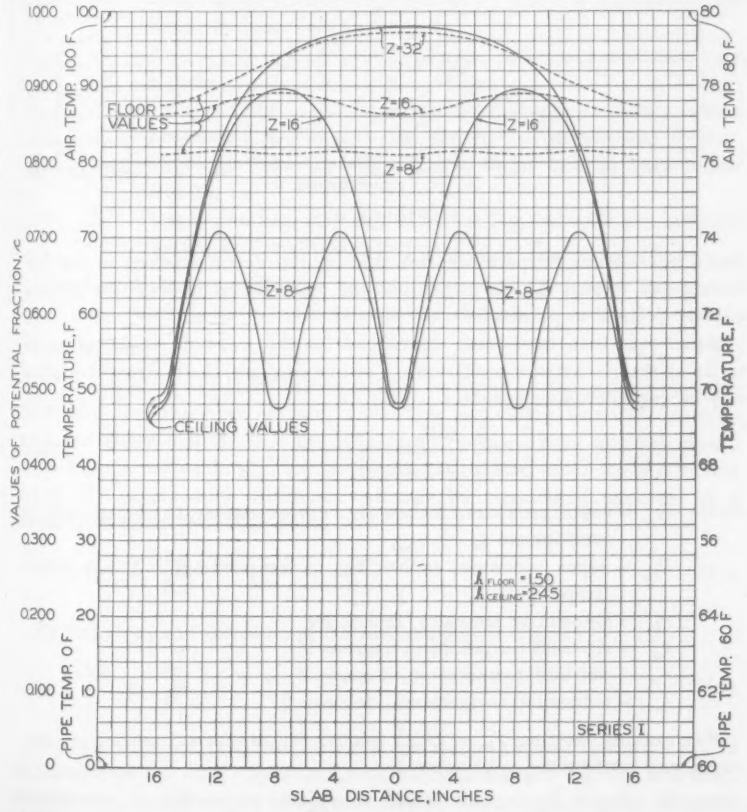

FIGURE 7.

FIGURE 6.

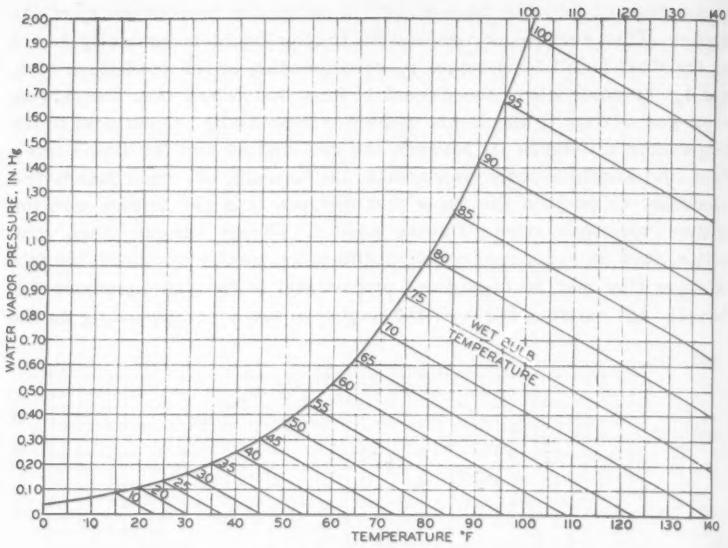

FIGURE 8.

liquid equilibrium temperature, and that for the vapor existent in the following gas stream. These conditions are represented in FIGURE 9 for a mixtute defined at point x.

Many equations have been developed for the wet-bulb temperature in terms of the air-mixture conditions. For the purpose, the Carrier[8] equation may be considered acceptable:

$$P_{t_s} = P_{t_w} - \left[\frac{P_b - P_{t_w}}{2830 - 1.44t_w}\right](t_d - t_w) \tag{10}$$

where P_{t_s} = vapor pressure, inches Hg, corresponding to the saturation temperature t_s

P_{t_w} = vapor pressure, inches Hg, corresponding to the wet-bulb temperature t_w

P_b = barometric pressure, inches Hg

t_d = dry-bulb temperature, degrees F.

t_w = wet-bulb temperature, degrees F.

t_s = saturation temperature, degrees F.

Rearranging, setting P_b = 29.92 (inches Hg standard barometric pressure), and introducing ratio-value F:

$$\frac{P_{t_w} - P_{t_s}}{t_d - t_w} = \frac{29.92 - P_{t_w}}{2830 - 1.44t_w} = F = \frac{\Delta P}{\Delta t} \tag{11}$$

From the above equation, it may be recognized that F is a function of the wet-bulb temperature t_w. Based on this equation, lines of constant wet-bulb temperature are shown in FIGURE 8. The value of F may be generalized as 0.01067 acceptably for normal ranges of air-conditioning operations.

Conditions of dynamic equilibrium are now to be considered:

For the heat transfer *to* the liquid: $q_u = Au\Delta t$ (12)

For the vapor movement *from* the liquid: $q_p = AK_p h_{fg} \Delta P$ (13)

where q_u = heat-transfer rate, Btu/hr.
A = liquid surface area, sq. ft.
u = heat-transfer conductance between air-mixture flow stream and liquid interface, Btu/(hr.-sq. ft.-degrees F.)
Δt = temperature difference degrees F. between air-mixture and water surface, that is, $(t_d - t_w)$
q_p = heat-flow rate associated with surface-evaporation rate, Btu/hr.
K_p = "Coefficient" of vapor diffusion under existing vapor-pressure difference, in lbs./(hr.-sq. ft.-inches Hg)
h_{fg} = Enthalpy value corresponding to latent heat of evaporation, Btu/lb.
ΔP = Vapor-pressure difference, inches Hg, between wet-bulb and saturation conditions, that is, $(P_{t_w} - P_{t_a})$

Under equilibrium conditions, as noted above, the heat-flow rates must equal each other (radiation effects assumed absent):

$$q_u = q_p \tag{14}$$

It is useful to consider the heat flow in terms of the resistance concept; that is, the heat transfer under the motivating force of the temperature difference Δt may be considered opposed by a thermal resistance R_u. Correspondingly, the *equivalent* heat flow under the motivating force of the pressure difference ΔP may be considered opposed by a vapor-flow resistance R_p. Thus,

$$q_u = \frac{\Delta t}{R_u} = q_p = \frac{\Delta P}{R_p} \tag{15}$$

Rearranging, and introducing the relationship of EQUATION 11:

$$\frac{\Delta P}{\Delta t} = \frac{R_p}{R_u} = F \tag{16}$$

It is to be noted that the boundary resistance to heat transfer, R_u, is dependent on the flow conditions past the heat transfer surface and that, furthermore, this boundary resistance may be associated with an *effective* "film" or layer between the main turbulent flow stream and the surface.

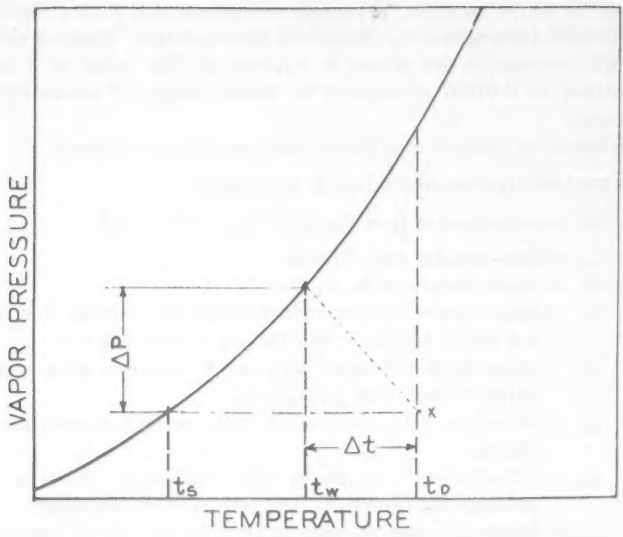

FIGURE 9.

This film, in turn, must be traversed by the exuding vapor; the two resistances R_u and R_p may then be considered definitely interconnected. This is acceptably substantiated by EQUATION 11, with its ratio value F. Therefore, R_p, may be shown as a function of R_u.

FIGURE 10 shows the energy-flow relationships for two cases, A and B, on a combination temperature and vapor-pressure versus resistance chart of range 0 – 100° F. Values for these two cases may conveniently be read off the psychrometric chart shown as FIGURE 8:

	t_d	t_w	P_t	t_s	P_{t_s}
Case A	100	75.0	0.875	64.5	0.611
Case B	80	60.0	0.522	45.1	0.310

The pairs of q lines in FIGURE 10 are parallel, certifying that $q_u = q_p$.

FIGURE 11 shows an electrical circuit arrangement carrying out the same idea as FIGURE 10, and includes a main circuit incorporating R_u and R_p. A temperature slide wire 0-100 and a vapor-pressure slide wire 0-2.0, scale ratio-factor 100/2 = 50, are also included: along with a source of direct current. The upper end of R_u is connected to a point on the temperature slide wire corresponding to t_d, and the lower end of R_p to a point on the vapor-pressure slide wire corresponding to the value P_{t_s} Adjusting resistance Z is manipulated progressively, with X and Y kept adjusted to satisfy their requirements, so that the lower end of R_u

and the upper end of R_p will give t_w and P_{t_w} values that coincide to give the right answer. Thus, if t_w were to read 75° F, P_{t_w} must simultaneously read 0.875 in. Hg. under conditions of galvanometer balance.

The previous circuit suggests the direct representation of the temperature relationships solely as a function of resistance. Using EQUATION 15 as the basis, a specialized and varying value of R_p may be developed, as R_p':

$$R_p' = \left[\frac{\Delta t_{ws}}{\Delta P}\right] FR_u \tag{17}$$

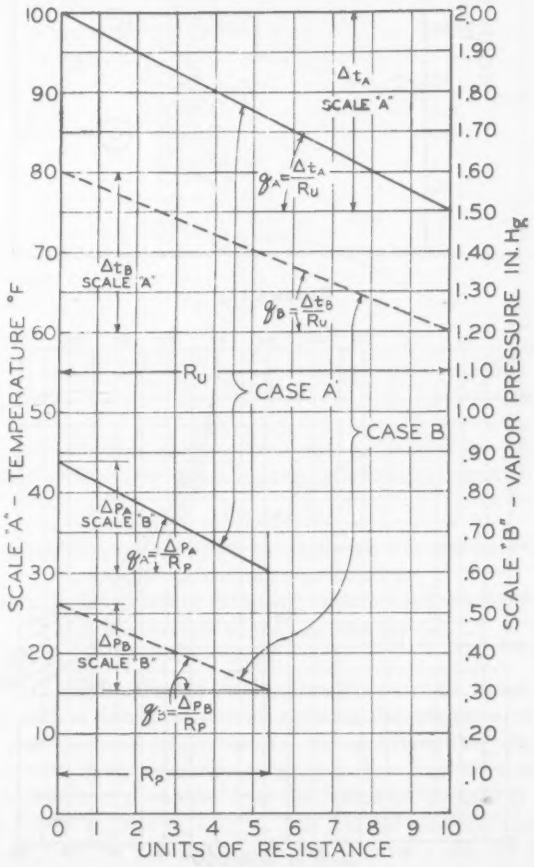

FIGURE 10.

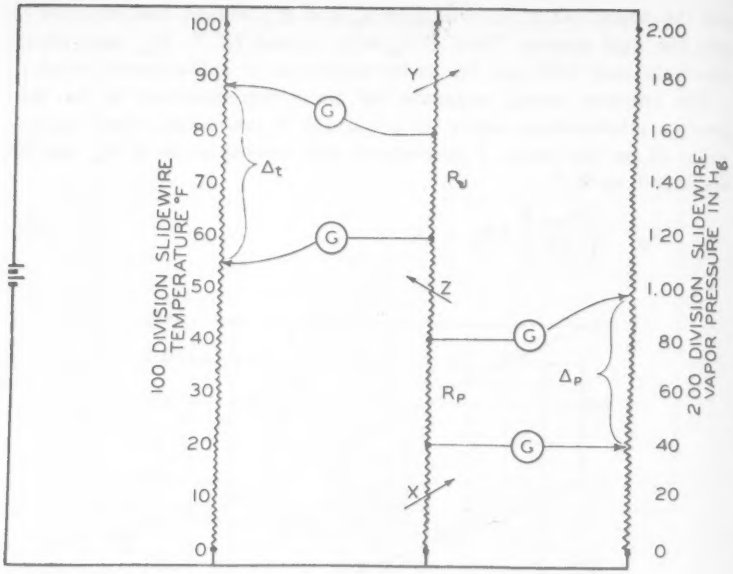

FIGURE 11.

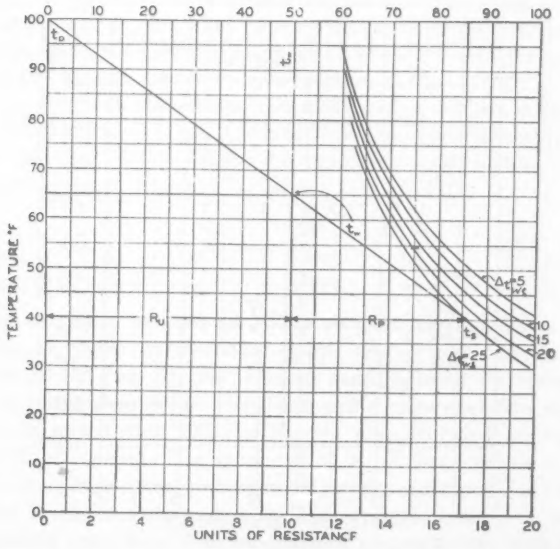

FIGURE 12.

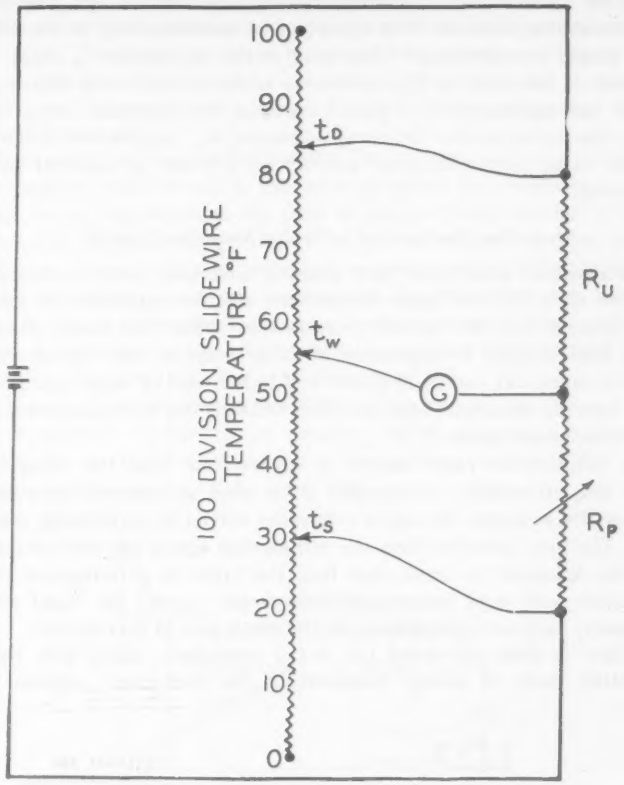

FIGURE 13.

where Δt_{ws} = temperature difference between the wet-bulb temperature t_w and the saturation temperature t_s

ΔP = vapor-pressure difference between the wet-bulb and saturation temperature vapor-pressure values;

F and R_u, as previously used.

FIGURE 12 shows the temperature-resistance chart based on EQUATION 17, with a family of curves showing the variation of R_p' under different conditions of Δt_{ws}, that is, the modifying $\Delta t_{ws}/\Delta P$ ratio not only varies over the temperature range, but also with the value of Δt_{ws}. The use of the chart is evident from the indicated example in which t_d = 100° F., t_s = 40° F., and t_w = 65° F. The straight line runs from 100 down to 40 at Δt_{ws} = 25, the selection of this latter value for Δt_{ws} being verified by the final result. The selection of the proper Δt_{ws} line is a matter of trial.

FIGURE 13 shows the circuit arrangement for determining t_w by electrical simulation methods. The upper end of resistance R_u is connected to the single "temperature" slide wire at the appropriate t_d value; the lower end of the variable R_p resistance is connected to the same slide wire at the appropriate t_s value. Observing the indicated Δt_{ws} value through the galvanometer balancing operation, R_p' may be set to the acceptable value on the basis of EQUATION 17, and t_w read off as the final result.

Heat-flow Analysis of a Cooled Structure Complex

In refrigerated structures, heat removal must take place continuously at a rate such that the space temperature may be maintained at its desired value below the outside temperature. Often the basic structure inward heat leakage is augmented by other sources, such as internally liberated electrical energy of power and lights, and of warm loads introduced from the outside. There may also be loads due to human occupancy and to ventilation needs.

The refrigeration plant serves to remove heat from the refrigerated space and, ultimately, to dissipate it to some atmospheric temperature receiver, for example, through a condenser served by circulating cooling water. The heat absorbed from the refrigerated space may be transferred from the space-air to brine, and from the brine to a refrigerant in an evaporator, and then transported through the agency of "heat pump" equipment, such as a compressor, to the condenser of this system. The heat flow is thus accounted for in the condenser, along with the refrigerating work of energy transport in the heat-pump machine. The

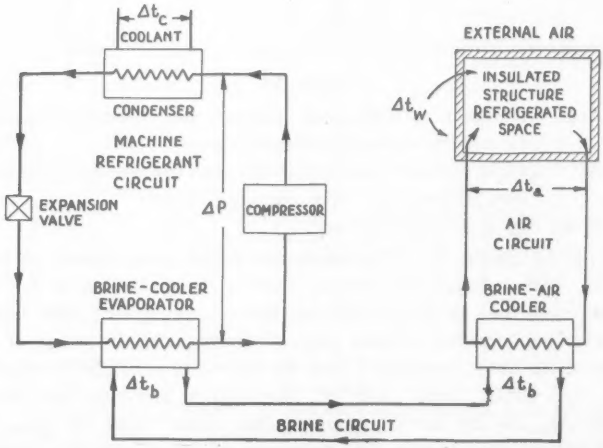

FIGURE 14.

particular problem, therefore, is one of predicting the over-all performance of the refrigerated structure, as associated with an integrated refrigeration plant. The latter includes its related heat-transfer equipment. Thus, the analysis is one devoted to a total complex process, in which the performance characteristics of all the individual components must be taken into account.[7]

FIGURE 14 represents the diagram for the interconnected elements of the complex, each of which can be represented by a resistance, to account for an opposition to the flow of energy. Thus, without expanding detail that is given elsewhere, it should be pointed out that the heat exchange processes are presented resistance-wise; the process of energy transport is similarly represented. For the latter, the energy involved traverses two appropriately valued resistances, one to take care of the energy pickup from the evaporator, and the other to take care of the energy required to do the work of transport.

FIGURE 15 shows the interrelation of the various temperature levels and variations for the entire complex, while FIGURE 16 shows the simulation electrical circuit for the computer to represent the cooled-structure complex. It will be noted that current meters are included, so

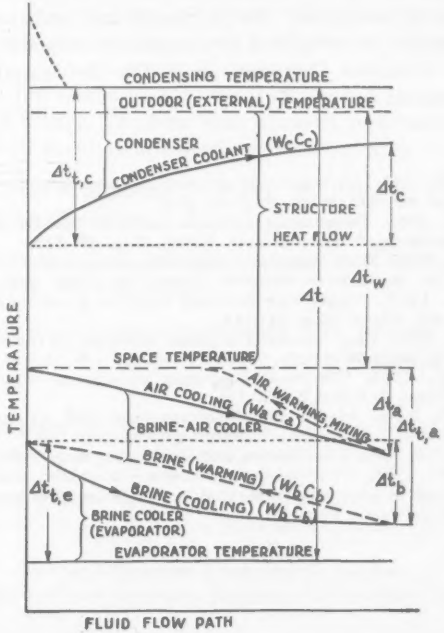

FIGURE 15.

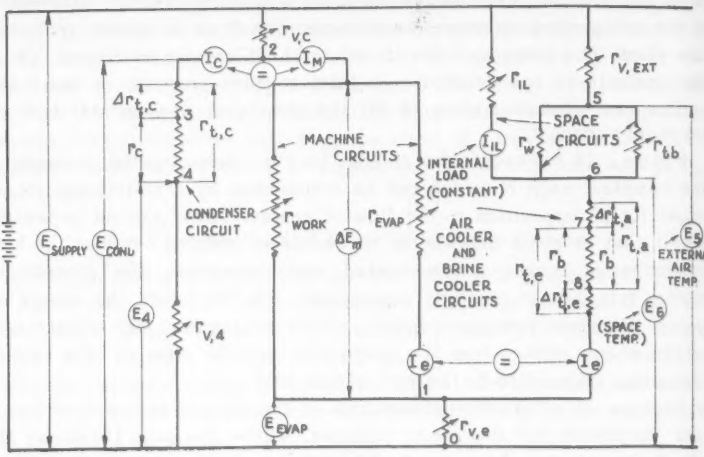

FIGURE 16.

that provision may be made to establish required equality of flow through different portions of the circuit, that is, equilibrium conditions.

This paper reports the results of investigations supported in part by a grant-in-aid to Columbia University from The Refrigeration Research Foundation, Colorado Springs, Colo.

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CONFERENCES HELD

SECTION OF BIOLOGY

BIOLOGICAL APPLICATIONS OF INFRARED SPECTROSCOPY

Friday, December 7 and
Saturday, December 8, 1956

Conference Co-Chairmen
Robert P. Bauman, Polytechnic Institute of Brooklyn, Brooklyn, N. Y.
Carl C. Clark, Naval Aviation Medical Acceleration Laboratory, Johnsville, Pa.

FRIDAY, DECEMBER 7, 1956

Session Chairman: Van Zandt Williams
Perkin-Elmer Corporation, Norwalk, Conn.

9:00 A.M. –
Greetings from the Academy – Frank C. Collins, Chairman, Section of Physics and Chemistry, The New York Academy of Sciences, New York, N.Y.; Polytechnic Institute of Brooklyn, Brooklyn, N. Y.

Introduction to the Conference – Robert P. Bauman, Polytechnic Institute of Brooklyn, Brooklyn, N. Y.

"The Place of Chemistry in Infrared Analysis" – R. C. Gore, American Cyanamid Company, Stamford, Conn.

"Utilization of Fractionation Procedures with Infrared Analysis" – Harris Rosenkrantz, Worcester Foundation for Experimental Biology, Shrewsbury, Mass.

"Automatic Analysis of Infrared Spectra" – Mortimer Rogoff, Federal Telecommunications Laboratories, Nutley, N. J.

"Intensity Measurements Applied to Determinations of Molecular Structure" – R. Norman Jones, National Research Council, Ottawa, Canada.

Session Chairman: Frederick C. Nachod
Sterling-Winthrop Research Institute, Rensselaer, N. Y.

2:00 P.M. –
"The Influence of Environment on Infrared Spectra" – Ralph Halford, Department of Chemistry, Columbia University, New York, N. Y.

"Some Simple Hydrogen Bonding Systems Studied by Infrared Absorption" – Urner Liddel, National Institute of Arthritis and Metabolic Diseases, National Institutes of Health, Bethesda, Md.

"Infrared Spectra of Biochemical Substances in Aqueous Solutions" – Elkan R. Blout, Polaroid Corporation, Cambridge, Mass.

"Infrared Studies of Amino Acids and Oligopeptides" – Robert J. Koegel, National Cancer Institute, National Institutes of Health, Bethesda, Md.

SATURDAY, DECEMBER 8, 1956

Session Chairman: Arthur W. Pollister
Columbia University, New York, N. Y.

9:00 A.M. –

"Infrared Studies of Tissue Lipids" – H. P. Schwarz, R. Childs, L. Dreisbach, and S. V. Mastrangelo, Philadelphia General Hospital, Philadelphia, Pa.

"Infrared Spectroscopy of Serum Lipids" – Norman K. Freeman, Donner Laboratory of Biophysics and Medical Physics, University of California, Berkeley, Calif.

"The Characterization of Mycobacterial Strains by Comparison of their Lipid Content" – Donald W. Smith and M. Gastambide-Odier, Department of Medical Microbiology, University of Wisconsin, Madison, Wis.; Harrison M. Randall and A. L. Koevoet, Harrison M. Randall Laboratory of Physics, University of Michigan, Ann Arbor, Mich.

"The Study of Virus Preparations by Infrared Spectroscopy" – A. A. Benedict, University of Texas – Medical Branch, Galveston, Tex.

Session Chairman: Lloyd C. Miller
U. S. Pharmacopeia, New York, N. Y.

2:00 P.M. –

"Infrared Studies of Tissues" – Leopold May and Robert G. Grenell, The Psychiatric Institute, University of Maryland, Baltimore, Md.

"Applications of Infrared Methods to Pharmaceutical Analysis" – Jonas Carol, Food and Drug Administration, Washington, D. C.

"Infrared Microspectroscopy in Biological Research" – Darwin L. Wood, Bell Telephone Laboratories, Murray Hill, N. J.

"The Availability of Infrared Spectra of Biochemicals" – Carl C. Clark, Naval Aviation Medical Acceleration Laboratory, Johnsville, Pa.

MODERN IDEAS ON SPONTANEOUS GENERATION

Wednesday, December 26, 1956

Conference Chairman: Ross F. Nigrelli
New York Zoological Society, Zoological Park
The Aquarium, New York, N. Y.

Session Chairman: Harold F. Blum
Princeton University, Princeton, N. J.

9:00 A.M. –

Greetings from the Academy – Hilary Koprowski, Chairman, Section of Biology, The New York Academy of Sciences, New York, N. Y.; Lederle Laboratories Division, American Cyanamid Company, Pearl River, N. Y.

"Formation of Organic Compounds on the Primitive Earth" – Stanley L. Miller, College of Physicians & Surgeons, Columbia University, New York, N. Y.

"Paleobiochemistry" – Philip H. Abelson, Carnegie Institution of Washington, Washington, D. C.

"Electrolytic Requirements of Protists and Archeo-Metabolism" – Seymour H. Hutner, Marvin Sanders, and J. J. A. McLaughlin, Haskins Laboratories, New York, N. Y.

"Speculations on Origins and Evolutions in Photosynthesis" – Sam Granick, Rockefeller Institute for Medical Research, New York, N. Y.

Session Chairman: George Wald
Harvard University, Cambridge, Mass.

2:00 P.M. –

"Phosphorous and the Origin of Life" – Addison Gulick, Cambridge, Mass.

"Interaction of Synthetic Polynucleotides" – Robert C. Warner, New York University College of Medicine, New York, N. Y.

"The Structure of Crystalline Proteins" – David Harker, Polytechnic Institute of Brooklyn, Brooklyn, N. Y.

"Spontaneous Generation of Protein and Anabolic Pathways" – Sidney W. Fox, The Florida State University, Tallahassee, Fla.

"The Gene as the Prime Mover" – Carl C. Lindegren, Southern Illinois University, Carbondale, Illinois.

Concluding Remarks – George Wald, Harvard University, Cambridge, Mass.

NEW MEMBERS

Elected December 20, 1956

SUSTAINING MEMBERSHIP

Farrington, J. D., M.D., Surgery. Assistant Professor, University of Illinois, Chicago, Ill.

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Gafafer, William M., D.Sc., Occupational Health, Adviser, U. S. Public Health Service, Washington, D. C.

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